Appendix D

Food Codes for NHANES

Table D.1 Food Codes for Leafy Produce

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
25	Spinach souffle	72125240
25	Broccoli casserole (broccoli, noodles, and cream sauce)	72202010
25	Broccoli casserole (broccoli, rice, cheese, and mushroom sau	72202020
25	Broccoli, batter-dipped and fried	72202030
25	Broccoli soup	72302000
25	Broccoli cheese soup, prepared with milk	72302100
25	Spinach soup	72307000
25	Dark-green leafy vegetable soup with meat, Oriental style	72308000
25	Dark-green leafy vegetable soup, meatless, Oriental style	72308500
25	Raw vegetable, NFS	75100250
25	Vegetables, NS as to type, cooked, NS as to fat added in coo	75200100
25	Vegetables, NS as to type, cooked, fat not added in cooking	75200110
25	Vegetable combination (including carrots, broccoli, and/or d	75440100
25	Vegetable tempura	75440200
25	Vegetables, dipped in chick-pea flour batter, (pakora), frie	75440400
25	Vegetable combinations (including carrots, broccoli, and/or	75440500
25	Vegetable combination (including carrots, broccoli, and/or d	75450500
25	Vegetable combinations (including carrots, broccoli, and/or	75460800
25	Vegetable soup, home recipe	75649110
25	Vegetable noodle soup, home recipe	75649150
25	Vegetable beef soup, home recipe	75652010
25	Vegetable beef soup with noodles or pasta, home recipe	75652040
25	Vegetable beef soup with rice, home recipe	75652050
33	Seven-layer salad (lettuce salad made with a combination of	75145000
33	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340110
33	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340120
50	Cabbage soup	75601200
50	Cabbage with meat soup	75601210
50	Broccoli and chicken, baby food, strained	76604000
75	Spinach, cooked, NS as to form, with cheese sauce	72125250
75	Turnip greens with roots, cooked, NS as to form, fat not add	72128410

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
75	Broccoli, cooked, NS as to form, with cheese sauce	72201230
75	Broccoli, cooked, from fresh, with cheese sauce	72201231
75	Broccoli, cooked, from frozen, with cheese sauce	72201232
75	Broccoli, cooked, NS as to form, with cream sauce	72201250
75	Broccoli, cooked, from fresh, with cream sauce	72201251
75	Cab age salad or coleslaw with apples and/or raisins, with dressing	75141100
75	Cabbage salad or coleslaw with pineapple, with dressing	75141200
75	Lettuce, salad with assorted vegetables including tomatoes a	75143000
75	Lettuce, salad with cheese, tomato and/or carrots, with or w	75143200
75	Lettuce salad with egg, cheese, tomato, and/or carrots, with	75143350
75	Spinach, creamed, baby food, strained	76102010
100	Beet greens, cooked, fat not added in cooking	72101210
100	Chard, cooked, fat not added in cooking	72104210
100	Chard, cooked, fat added in cooking	72104220
100	Collards, raw	72107100
100	Collards, cooked, NS as to form, NS as to fat added in cooki	72107200
100	Collards, cooked, from fresh, NS as to fat added in cooking	72107201
100	Collards, cooked, from fresh, fat not added in cooking	72107211
100	Collards, cooked, NS as to form, fat added in cooking	72107220
100	Collards, cooked, from fresh, fat added in cooking	72107221
100	Collards, cooked, from frozen, fat added in cooking	72107222
100	Greens, cooked, from fresh, fat not added in cooking	72118211
100	Greens, cooked, NS as to form, fat added in cooking	72118220
100	Greens, cooked, from fresh, fat added in cooking	72118221
100	Kale, cooked, NS as to form, NS as to fat added in cooking	72119200
100	Kale, cooked, from fresh, fat not added in cooking	72119211
100	Kale, cooked, NS as to form, fat added in cooking	72119220
100	Kale, cooked, from fresh, fat added in cooking	72119221
100	Mustard greens, cooked, NS as to form, NS as to fat added in	72122200
100	Mustard greens, cooked, from fresh, NS as to fat added in co	72122201
100	Mustard greens, cooked, from fresh, fat not added in cooking	72122211
100	Mustard greens, cooked, from canned, fat not added in cookin	72122213

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
100	Mustard greens, cooked, from fresh, fat added in cooking	72122221
100	Mustard greens, cooked, from frozen, fat added in cooking	72122222
100	Mustard greens, cooked, from canned, fat added in cooking	72122223
100	Poke greens, cooked, fat not added in cooking	72123010
100	Poke greens, cooked, fat added in cooking	72123020
100	Radicchio, raw	72124100
100	Spinach, raw	72125100
100	Spinach, cooked, NS as to form, NS as to fat added in cookin	72125200
100	Spinach, cooked, from fresh, NS as to fat added in cooking	72125201
100	Spinach, cooked, from frozen, NS as to fat added in cooking	72125202
100	Spinach, cooked, NS as to form, fat not added in cooking	72125210
100	Spinach, cooked, from fresh, fat not added in cooking	72125211
100	Spinach, cooked, from frozen, fat not added in cooking	72125212
100	Spinach, cooked, NS as to form, fat added in cooking	72125220
100	Spinach, cooked, from fresh, fat added in cooking	72125221
100	Spinach, cooked, from frozen, fat added in cooking	72125222
100	Spinach, NS as to form, creamed	72125230
100	Turnip greens, cooked, from fresh, fat not added in cooking	72128211
100	Turnip greens, cooked, NS as to form, fat added in cooking	72128220
100	Turnip greens, cooked, from fresh, fat added in cooking	72128221
100	Turnip greens, cooked, from frozen, fat added in cooking	72128222
100	Watercress, raw	72130100
100	Broccoli, raw	72201100
100	Broccoli, cooked, NS as to form, NS as to fat added in cooki	72201200
100	Broccoli, cooked, from fresh, NS as to fat added in cooking	72201201
100	Broccoli, cooked, from frozen, NS as to fat added in cooking	72201202
100	Broccoli, cooked, NS as to form, fat not added in cooking	72201210
100	Broccoli, cooked, from fresh, fat not added in cooking	72201211
100	Broccoli, cooked, from frozen, fat not added in cooking	72201212
100	Broccoli, cooked, NS as to form, fat added in cooking	72201220
100	Broccoli, cooked, from fresh, fat added in cooking	72201221
100	Broccoli, cooked, from frozen, fat added in cooking	72201222

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
100	Sprouts, NFS	75100300
100	Alfalfa sprouts, raw	75100500
100	Artichoke, Jerusalem, raw	75100750
100	Cabbage, green, raw	75103000
100	Cabbage, Chinese, raw	75104000
100	Cabbage, red, raw	75105000
100	Cauliflower, raw	75107000
100	Celery, raw	75109000
100	Chives, raw	75109500
100	Cilantro, raw	75109550
100	Lettuce, raw	75113000
100	Lettuce, Boston, raw	75113060
100	Lettuce, arugula, raw	75113080
100	Mixed salad greens, raw	75114000
100	Parsley, raw	75119000
100	Broccoli salad with cauliflower, cheese, bacon bits, and dre	75140500
100	Cabbage salad or coleslaw, with dressing	75141000
100	Artichoke, globe (French), cooked, NS as to form, NS as to f	75201000
100	Artichoke, globe (French), cooked, NS as to form, fat not ad	75201010
100	Artichoke, globe (French), cooked, from fresh, fat not added	75201011
100	Artichoke, globe (French), cooked, from canned, fat not adde	75201013
100	Artichoke, globe (French), cooked, NS as to form, fat added	75201020
100	Artichoke, globe (French), cooked, from fresh, fat added in	75201021
100	Artichoke salad in oil	75201030
100	Brussels sprouts, cooked, NS as to form, fat not added in co	75209010
100	Brussels sprouts, cooked, from fresh, fat not added in cooki	75209011
100	Brussels sprouts, cooked, from frozen, fat not added in cook	75209012
100	Brussels sprouts, cooked, from fresh, fat added in cooking	75209021
100	Brussels sprouts, cooked, from frozen, fat added in cooking	75209022
100	Cabbage, Chinese, cooked, NS as to fat added in cooking	75210000
100	Cabbage, Chinese, cooked, fat not added in cooking	75210010
100	Cabbage, Chinese, cooked, fat added in cooking	75210020

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
100	Cabbage, green, cooked, NS as to fat added in cooking	75211010
100	Cabbage, green, cooked, fat not added in cooking	75211020
100	Cabbage, green, cooked, fat added in cooking	75211030
100	Cabbage, red, cooked, fat not added in cooking	75212010
100	Cauliflower, cooked, NS as to form, NS as to fat added in co	75214000
100	Cauliflower, cooked, from fresh, NS as to fat added in cooki	75214001
100	Cauliflower, cooked, from frozen, NS as to fat added in cook	75214002
100	Cauliflower, cooked, NS as to form, fat not added in cooking	75214010
100	Cauliflower, cooked, from fresh, fat not added in cooking	75214011
100	Cauliflower, cooked, from frozen, fat not added in cooking	75214012
100	Cauliflower, cooked, NS as to form, fat added in cooking	75214020
100	Cauliflower, cooked, from fresh, fat added in cooking	75214021
100	Cauliflower, cooked, from frozen, fat added in cooking	75214022
100	Lettuce, cooked, fat not added in cooking	75220050
100	Parsley, cooked (assume fat not added in cooking)	75221210
100	Cauliflower, batter-dipped, fried	75409020
100	Cabbage, red, pickled	75502510
100	Cabbage, Kim Chee style	75502520

Table D.2 Food Codes for Exposed Produce

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
12.5	Vegetable beef soup, home recipe	75652010
12.5	Vegetable beef soup with noodles or pasta, home recipe	75652040
12.5	Vegetable beef soup with rice, home recipe	75652050
12.5	Vegetables and rice, baby food, strained	76501000
12.5	Vegetable and bacon, baby food, strained	76601010
12.5	Vegetable and beef, baby food, strained	76603010
12.5	Vegetable and beef, baby food, junior	76603020
12.5	Vegetable and chicken, baby food, strained	76605010
12.5	Vegetable and chicken, baby food, junior	76605020
12.5	Vegetable and ham, baby food, strained	76607010
12.5	Vegetable and ham, baby food, junior	76607020
12.5	Vegetable and turkey, baby food, strained	76611010
12.5	Vegetable and turkey, baby food, junior	76611020
25.0	Raw vegetable, NFS	75100250
25.0	Cabbage salad or coleslaw with apples and/or raisins, with d	75141100
25.0	Vegetables, NS as to type, cooked, NS as to fat added in coo	75200100
25.0	Vegetables, NS as to type, cooked, fat not added in cooking	75200110
25.0	Vegetable combination (including carrots, broccoli, and/or d	75440100
25.0	Vegetable tempura	75440200
25.0	Vegetable combinations (including carrots, broccoli, and/or	75440500
25.0	Vegetable combination (including carrots, broccoli, and/or d	75450500
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460800
25.0	Vegetable soup, home recipe	75649110
25.0	Vegetable noodle soup, home recipe	75649150
25.0	Spanish stew, Puerto Rican style (Cocido Espanol)	77513010
33.0	Grape juice	64116020
33.0	Peach juice, with sugar	64122030
33.0	Apple-banana juice, baby food	67203200
33.0	Apple-cranberry juice, baby food	67203450
33.0	Tomato soup, NFS	74601000

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
33.0	Tomato soup, prepared with water	74602010
33.0	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340110
33.0	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340120
33.0	Vegetable stew without meat	75439010
33.0	Mushroom soup, NFS	75607000
33.0	Mixed vegetables, garden vegetables, baby food, NS as to str	76407000
33.0	Mixed vegetables, garden vegetables, baby food, strained	76407010
33.0	Mixed vegetables, garden vegetables, baby food, junior	76407020
33.0	Jams, preserves, marmalades, dietetic, all flavors, sweetene	91406000
33.0	Jams, preserves, marmalades, sweetened with fruit juice conc	91406500
33.0	Jams, preserves, marmalades, low sugar (all flavors)	91406600
50.0	Bananas with apples and pears, baby food, strained	67106010
50.0	Pears and pineapple, baby food, strained	67114010
50.0	Pears and pineapple, baby food, junior	67114020
50.0	Tomato and corn, cooked, fat not added in cooking	74503010
50.0	Tomato and onion, cooked, NS as to fat added in cooking	74504100
50.0	Tomato and onion, cooked, fat not added in cooking	74504110
50.0	Tomato and onion, cooked, fat added in cooking	74504120
50.0	Beans, green, and potatoes, cooked, fat not added in cooking	75302050
50.0	Beans, green, with pinto beans, cooked, fat not added in coo	75302060
50.0	Beans, green, and potatoes, cooked, NS as to fat added in co	75302500
50.0	Beans, green, and potatoes, cooked, fat added in cooking	75302510
50.0	Peas with mushrooms, cooked, fat not added in cooking	75315210
50.0	Chiles rellenos, cheese-filled (stuffed chili peppers)	75410500
50.0	Chiles rellenos, filled with meat and cheese (stuffed chili	75410530
50.0	Minestrone soup, home recipe	75651000
50.0	Jelly, all flavors	91401000
50.0	Jam, preserves, all flavors	91402000
50.0	Jelly, dietetic, all flavors, sweetened with artificial swee	91405000
50.0	Jelly, reduced sugar, all flavors	91405500
66.0	Fruit juice, NFS	64100100
66.0	Apple cider	64101010

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
66.0	Apple juice	64104010
66.0	Prune juice	64132010
66.0	Prune juice, unsweetened	64132020
66.0	Strawberry juice	64132500
66.0	Apple juice, baby food	67202000
66.0	Apple with other fruit juice, baby food	67203000
66.0	Apple-cherry juice, baby food	67203400
66.0	Apple-grape juice, baby food	67203500
66.0	Apple-prune juice, baby food	67203700
66.0	Grape juice, baby food	67203800
66.0	Mixed fruit juice, not citrus, baby food	67204000
66.0	Pear juice, baby food	67212000
66.0	Tomato juice	74301100
66.0	Tomato and vegetable juice, mostly tomato	74303000
66.0	Mixed vegetable juice (vegetables other than tomato)	75132000
66.0	Celery juice	75132100
66.0	Gazpacho	75604600
100.0	Fruit, dried, NFS (assume uncooked)	62101000
100.0	Fruit mixture, dried (mixture includes three or more of the	62101050
100.0	Apple, dried, uncooked	62101100
100.0	Apple, dried, cooked, NS as to sweetened or unsweetened; swe	62101200
100.0	Apricot, dried, uncooked	62104100
100.0	Pear, dried, cooked, with sugar	62119230
100.0	Prune, dried, uncooked	62122100
100.0	Prune, dried, cooked, NS as to sweetened or unsweetened; swe	62122200
100.0	Prune, dried, cooked, unsweetened	62122220
100.0	Prune, dried, cooked, with sugar	62122230
100.0	Raisins	62125100
100.0	Raisins, cooked	62125110
100.0	Apple, raw	63101000
100.0	Applesauce, stewed apples, NS as to sweetened or unsweetened	63101110
100.0	Applesauce, stewed apples, unsweetened	63101120

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
100.0	Applesauce, stewed apples, with sugar	63101130
100.0	Applesauce, stewed apples, sweetened with low calorie sweete	63101140
100.0	Applesauce with other fruits	63101150
100.0	Apple, cooked or canned, with syrup	63101210
100.0	Apple, baked, NS as to added sweetener	63101310
100.0	Apple, baked, unsweetened	63101320
100.0	Apple, baked, with sugar	63101330
100.0	Apple, pickled	63101420
100.0	Apple, fried	63101500
100.0	Apricot, raw	63103010
100.0	Apricot, cooked or canned, NS as to sweetened or unsweetened	63103110
100.0	Apricot, cooked or canned, in light syrup	63103140
100.0	Apricot, cooked or canned, drained solids	63103150
100.0	Apricot, cooked or canned, juice pack	63103170
100.0	Cherry pie filling	63113030
100.0	Cherries, sweet, raw (Queen Anne, Bing)	63115010
100.0	Cherries, sweet, cooked or canned, drained solids	63115150
100.0	Fig, raw	63119010
100.0	Grapes, raw, NS as to type	63123000
100.0	Grapes, European type, adherent skin, raw	63123010
100.0	Grapes, seedless, cooked or canned, unsweetened, water pack	63123120
100.0	Mango, raw	63129010
100.0	Mango, cooked	63129030
100.0	Nectarine, raw	63131010
100.0	Nectarine, cooked	63131110
100.0	Peach, raw	63135010
100.0	Peach, cooked or canned, NS as to sweetened or unsweetened;	63135110
100.0	Peach, cooked or canned, in heavy syrup	63135130
100.0	Peach, cooked or canned, in light or medium syrup	63135140
100.0	Peach, cooked or canned, drained solids	63135150
100.0	Peach, cooked or canned, juice pack	63135170
100.0	Peach, frozen, NS as to added sweetener	63135610

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
100.0	Peach, frozen, unsweetened	63135620
100.0	Peach, frozen, with sugar	63135630
100.0	Pear, raw	63137010
100.0	Pear, Japanese, raw	63137050
100.0	Pear, cooked or canned, NS as to sweetened or unsweetened; s	63137110
100.0	Pear, cooked or canned, in heavy syrup	63137130
100.0	Pear, cooked or canned, in light syrup	63137140
100.0	Pear, cooked or canned, drained solids	63137150
100.0	Pear, cooked or canned, juice pack	63137170
100.0	Persimmon, raw	63139010
100.0	Plum, raw	63143010
100.0	Plum, cooked or canned, in light syrup	63143140
100.0	Plum, pickled	63143650
100.0	Rhubarb, frozen, with sugar	63147620
100.0	SUGAR APPLE, SWEETSOP (ANON), RAW	63148010
100.0	Blackberries, raw	63201010
100.0	Blackberries, cooked or canned, NS as to sweetened or unswee	63201110
100.0	Raspberries, raw, NS as to color	63219000
100.0	Raspberries, red, raw	63219020
100.0	Raspberries, cooked or canned, NS as to sweetened or unsweet	63219110
100.0	Raspberries, frozen, unsweetened	63219610
100.0	Strawberries, raw	63223020
100.0	Strawberries, raw, with sugar	63223030
100.0	Strawberries, cooked or canned, NS as to sweetened or unswee	63223110
100.0	Strawberries, cooked or canned, unsweetened, water pack	63223120
100.0	Strawberries, cooked or canned, in syrup	63223130
100.0	Strawberries, frozen, NS as to added sweetener	63223600
100.0	Strawberries, frozen, unsweetened	63223610
100.0	Strawberries, frozen, with sugar	63223620
100.0	Fruit cocktail or mix (excluding citrus fruits), raw	63311000
100.0	Apple salad with dressing	63401010
100.0	Apple, candied	63401060

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
100.0	Fruit salad (excluding citrus fruits) with salad dressing or	63402950
100.0	Fruit salad (excluding citrus fruits) with cream	63402960
100.0	Fruit salad (excluding citrus fruits) with cream substitute	63402970
100.0	Fruit salad (excluding citrus fruits) with marshmallows	63402980
100.0	Fruit salad (excluding citrus fruits) with pudding	63403000
100.0	Fruit salad (including citrus fruits) with salad dressing or	63403010
100.0	Fruit salad (including citrus fruit) with cream	63403020
100.0	Fruit salad (including citrus fruits) with marshmallows	63403040
100.0	Chutney	63409020
100.0	Tomato and okra, cooked, NS as to fat added in cooking	74504000
100.0	Tomato and okra, cooked, fat not added in cooking	74504010
100.0	Tomato and okra, cooked, fat added in cooking	74504020
100.0	Tomato and celery, cooked, fat not added in cooking	74504150
100.0	Cucumber salad with creamy dressing	75142500
100.0	Cucumber salad made with cucumber, oil, and vinegar	75142550
100.0	Cucumber salad made with cucumber and vinegar	75142600
100.0	Cucumber pickles, dill	75503010
100.0	Cucumber pickles, relish	75503020
100.0	Cucumber pickles, sour	75503030
100.0	Cucumber pickles, sweet	75503040
100.0	Cucumber pickles, fresh	75503050
100.0	Mustard pickles	75503100
100.0	Cucumber pickles, dill, reduced salt	75503110

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
12.5	Vegetables and rice, baby food, strained	76501000
12.5	Vegetable and bacon, baby food, strained	76601010
12.5	Carrots and beef, baby food, strained	76602000
12.5	Vegetable and beef, baby food, strained	76603010
12.5	Vegetable and beef, baby food, junior	76603020
12.5	Vegetable and chicken, baby food, strained	76605010
12.5	Vegetable and chicken, baby food, junior	76605020
12.5	Vegetable and ham, baby food, strained	76607010
12.5	Vegetable and ham, baby food, junior	76607020
12.5	Vegetable and turkey, baby food, strained	76611010
12.5	Vegetable and turkey, baby food, junior	76611020
25.0	Lemon pie filling	61113500
25.0	Vegetables, NS as to type, cooked, NS as to fat added in coo	75200100
25.0	Vegetables, NS as to type, cooked, fat not added in cooking	75200110
25.0	Vegetable combination (including carrots, broccoli, and/or d	75440100
25.0	Vegetable combination (excluding carrots, broccoli, and dark	75440110
25.0	Vegetable sticks, breaded (including corn, carrots, and gree	75440170
25.0	Vegetable tempura	75440200
25.0	Vegetables, dipped in chick-pea flour batter, (pakora), frie	75440400
25.0	Vegetable combinations (including carrots, broccoli, and/or	75440500
25.0	Vegetable combination (including carrots, broccoli, and/or d	75450500
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460700
25.0	Vegetable combinations (excluding carrots, broccoli, and dar	75460710
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460800
25.0	Vegetable soup, home recipe	75649110
25.0	Vegetable noodle soup, home recipe	75649150
25.0	Vegetable beef soup, home recipe	75652010
25.0	Vegetable beef soup with noodles or pasta, home recipe	75652040
25.0	Vegetable beef soup with rice, home recipe	75652050
25.0	Fruit sauce	91361020

% Protected Produce in Food Item	Food Item Description	USDA Food Code
33.0	Strawberry-banana-orange juice	61226000
33.0	Vegetable stew without meat	75439010
33.0	Mixed vegetables, garden vegetables, baby food, NS as to str	76407000
33.0	Mixed vegetables, garden vegetables, baby food, strained	76407010
33.0	Mixed vegetables, garden vegetables, baby food, junior	76407020
33.0	Jams, preserves, marmalades, dietetic, all flavors, sweetene	91406000
33.0	Jams, preserves, marmalades, sweetened with fruit juice conc	91406500
33.0	Jams, preserves, marmalades, low sugar (all flavors)	91406600
50.0	Orange and banana juice	61219000
50.0	Pineapple-orange juice, NFS	61225000
50.0	Tomato and corn, cooked, fat not added in cooking	74503010
50.0	Beans, green, with pinto beans, cooked, fat not added in coo	75302060
50.0	Peas and onions, cooked, fat not added in cooking	75315110
50.0	Peas and onions, cooked, fat added in cooking	75315120
50.0	Peas with mushrooms, cooked, fat not added in cooking	75315210
50.0	Peas and potatoes, cooked, fat not added in cooking	75315300
50.0	Squash, summer, and onions, cooked, fat not added in cooking	75316000
50.0	Pinacbet (eggplant with tomatoes, bitter melon, etc.)	75340300
50.0	Eggplant, batter-dipped, fried	75412010
50.0	Eggplant dip	75412030
50.0	Eggplant parmesan casserole, regular	75412060
50.0	Pea salad	75416500
50.0	Pea salad with cheese	75416600
50.0	Squash,summer, yellow or green, breaded or battered, baked	75418000
50.0	Squash, summer, yellow or green, breaded or battered, fried	75418010
50.0	Pea soup, NFS	75609000
50.0	Carrots and peas, baby food, strained	76202000
100.0	Almonds, NFS	42100100
100.0	Almonds, unroasted	42101000
100.0	Chestnuts, roasted	42105000
100.0	Filberts, hazelnuts	42107000
100.0	Pecans	42112000

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Walnuts	42116000
100.0	Pumpkin and/or squash seeds, hulled, roasted, salted	43101100
100.0	Grapefruit, raw	61101010
100.0	Grapefruit, canned or frozen, NS as to sweetened or unsweete	61101200
100.0	Grapefruit, canned or frozen, in light syrup	61101230
100.0	Lemon, raw	61113010
100.0	Lime, raw	61116010
100.0	Orange, raw	61119010
100.0	Orange, mandarin, canned or frozen, NS as to sweetened or un	61122300
100.0	Orange, mandarin, canned or frozen, juice pack	61122320
100.0	Orange, mandarin, canned or frozen, in light syrup	61122330
100.0	Orange, mandarin, canned or frozen, drained	61122350
100.0	Tangerine, raw	61125010
100.0	Grapefruit juice, freshly squeezed	61201010
100.0	Lemon juice, NS as to form	61204000
100.0	Lemon juice, fresh	61204010
100.0	Lemon juice, frozen	61204600
100.0	Lime juice, NS as to form	61207000
100.0	Lime juice, fresh	61207010
100.0	Lime juice, frozen	61207600
100.0	Orange juice, NFS	61210000
100.0	Orange juice, freshly squeezed	61210010
100.0	Tangerine juice, NFS	61213000
100.0	Avocado, raw	63105010
100.0	Cantaloupe (muskmelon), raw	63109010
100.0	Cantaloupe, frozen (balls)	63109610
100.0	Kiwi fruit, raw	63126500
100.0	Honeydew melon, raw	63127010
100.0	Honeydew, frozen (balls)	63127610
100.0	Papaya, raw	63133010
100.0	Papaya, cooked or canned, in sugar or syrup	63133100
100.0	Pomegranate, raw	63145010

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Watermelon, raw	63149010
100.0	Guacamole with tomatoes	63408010
100.0	Guacamole with tomatoes and chili peppers	63408200
100.0	Guacamole, NFS	63409010
100.0	Pumpkin, cooked, from fresh, fat not added in cooking	73201011
100.0	Pumpkin, cooked, from canned, fat not added in cooking	73201013
100.0	Pumpkin, cooked, NS as to form, fat added in cooking	73201020
100.0	Pumpkin, cooked, from fresh, fat added in cooking	73201021
100.0	Calabaza (Spanish pumpkin), cooked	73210010
100.0	Squash, winter type, mashed, NS as to fat or sugar added in	73301000
100.0	Squash, winter type, mashed, no fat or sugar added in cookin	73301010
100.0	Squash, winter type, mashed, fat added in cooking, no sugar	73301020
100.0	Squash, winter type, baked, NS as to fat or sugar added in c	73303000
100.0	Squash, winter type, baked, no fat or sugar added in cooking	73303010
100.0	Squash, winter type, baked, fat added in cooking, no sugar a	73303020
100.0	Squash, winter, baked with cheese	73305010
100.0	Peas, green, raw	75120000
100.0	Squash, summer, yellow, raw	75128000
100.0	Squash, summer, green, raw	75128010
100.0	Beans, lima, immature, cooked, NS as to form, NS as to fat a	75204000
100.0	Beans, lima, immature, cooked, from fresh, fat not added in	75204011
100.0	Beans, lima, immature, cooked, from frozen, fat not added in	75204012
100.0	Beans, lima, immature, cooked, NS as to form, fat added in c	75204020
100.0	Beans, lima, immature, cooked, from fresh, fat added in cook	75204021
100.0	Beans, lima, immature, cooked, from frozen, fat added in coo	75204022
100.0	Bitter melon, cooked, fat added in cooking	75208310
100.0	Cactus, cooked, NS as to fat added in cooking	75213100
100.0	Cactus, cooked, fat not added in cooking	75213110
100.0	Cactus, cooked, fat added in cooking	75213120
100.0	Christophine, cooked, fat not added in cooking	75215510
100.0	Corn, cooked, NS as to form, NS as to color, NS as to fat ad	75216000
100.0	Corn, cooked, from fresh, NS as to color, NS as to fat added	75216001

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Corn, cooked, from frozen, NS as to color, NS as to fat adde	75216002
100.0	Corn, cooked, NS as to form, NS as to color, fat not added i	75216010
100.0	Corn, cooked, from fresh, NS as to color, fat not added in c	75216011
100.0	Corn, cooked, from frozen, NS as to color, fat not added in	75216012
100.0	Corn, cooked, NS as to form, NS as to color, fat added in co	75216020
100.0	Corn, cooked, from fresh, NS as to color, fat added in cooki	75216021
100.0	Corn, cooked, from frozen, NS as to color, fat added in cook	75216022
100.0	Corn, NS as to form, NS as to color, cream style	75216050
100.0	Corn, yellow, cooked, NS as to form, NS as to fat added in c	75216100
100.0	Corn, yellow, cooked, from fresh, NS as to fat added in cook	75216101
100.0	Corn, yellow, cooked, from frozen, NS as to fat added in coo	75216102
100.0	Corn, yellow, cooked, NS as to form, fat not added in cookin	75216110
100.0	Corn, yellow, cooked, from fresh, fat not added in cooking	75216111
100.0	Corn, yellow, cooked, from frozen, fat not added in cooking	75216112
100.0	Corn, yellow, cooked, NS as to form, fat added in cooking	75216120
100.0	Corn, yellow, cooked, from fresh, fat added in cooking	75216121
100.0	Corn, yellow, cooked, from frozen, fat added in cooking	75216122
100.0	Corn, yellow, NS as to form, cream style	75216150
100.0	Corn, yellow and white, cooked, NS as to form, NS as to fat	75216160
100.0	Corn, yellow and white, cooked, from fresh, NS as to fat add	75216161
100.0	Corn, yellow and white, cooked, NS as to form, fat not added	75216170
100.0	Corn, yellow and white, cooked, from fresh, fat not added in	75216171
100.0	Corn, yellow and white, cooked, from fresh, fat added in coo	75216181
100.0	Corn, white, cooked, NS as to form, NS as to fat added in co	75216200
100.0	Corn, white, cooked, from fresh, NS as to fat added in cooki	75216201
100.0	Corn, white, cooked, NS as to form, fat not added in cooking	75216210
100.0	Corn, white, cooked, from fresh, fat not added in cooking	75216211
100.0	Corn, white, cooked, from frozen, fat not added in cooking	75216212
100.0	Corn, white, cooked, from fresh, fat added in cooking	75216221
100.0	Corn, white, cooked, from frozen, fat added in cooking	75216222
100.0	Hominy, cooked, fat not added in cooking	75217500
100.0	Hominy, cooked, fat added in cooking	75217520

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Peas, cowpeas, field peas, or blackeye peas (not dried), coo	75223000
100.0	Peas, cowpeas, field peas, or blackeye peas (not dried), coo	75223020
100.0	Peas, cowpeas, field peas, or blackeye peas (not dried), coo	75223021
100.0	Peas, cowpeas, field peas, or blackeye peas (not dried), coo	75223022
100.0	Peas, green, cooked, NS as to form, NS as to fat added in co	75224010
100.0	Peas, green, cooked, from fresh, NS as to fat added in cooki	75224011
100.0	Peas, green, cooked, from frozen, NS as to fat added in cook	75224012
100.0	Peas, green, cooked, NS as to form, fat not added in cooking	75224020
100.0	Peas, green, cooked, from fresh, fat not added in cooking	75224021
100.0	Peas, green, cooked, from frozen, fat not added in cooking	75224022
100.0	Peas, green, cooked, NS as to form, fat added in cooking	75224030
100.0	Peas, green, cooked, from fresh, fat added in cooking	75224031
100.0	Peas, green, cooked, from frozen, fat added in cooking	75224032
100.0	Pigeon peas, cooked, NS as to form, fat not added in cooking	75225010
100.0	Squash, summer, cooked, NS as to form, NS as to fat added in	75233000
100.0	Squash, summer, cooked, from fresh, NS as to fat added in co	75233001
100.0	Squash, summer, cooked, from frozen, NS as to fat added in c	75233002
100.0	Squash, summer, cooked, NS as to form, fat not added in cook	75233010
100.0	Squash, summer, cooked, from fresh, fat not added in cooking	75233011
100.0	Squash, summer, cooked, from frozen, fat not added in cookin	75233012
100.0	Squash, summer, cooked, NS as to form, fat added in cooking	75233020
100.0	Squash, summer, cooked, from fresh, fat added in cooking	75233021
100.0	Beans, lima and corn (succotash), cooked, fat not added in c	75301110
100.0	Beans, lima and corn (succotash), cooked, fat added in cooki	75301120
100.0	Peas and corn, cooked, NS as to fat added in cooking	75315000
100.0	Peas and corn, cooked, fat not added in cooking	75315010
100.0	Peas and corn, cooked, fat added in cooking	75315020
100.0	Squash, baby food, strained	76205010
100.0	Corn, creamed, baby food, strained	76405010
100.0	Corn, creamed, baby food, junior	76405020
100.0	Peas, baby food, NS as to strained or junior	76409000
100.0	Peas, baby food, strained	76409010

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Peas, baby food, junior	76409020
100.0	Marmalade, all flavors	91404000
12.5	Beet soup (borscht)	75601100
12.5	Leek soup, cream of, prepared with milk	75605010
12.5	Onion soup, French	75608100
12.5	Vegetables and rice, baby food, strained	76501000
12.5	Vegetable and bacon, baby food, strained	76601010
12.5	Vegetable and beef, baby food, strained	76603010
12.5	Vegetable and beef, baby food, junior	76603020
12.5	Vegetable and chicken, baby food, strained	76605010
12.5	Vegetable and chicken, baby food, junior	76605020
12.5	Vegetable and ham, baby food, strained	76607010
12.5	Vegetable and ham, baby food, junior	76607020
12.5	Vegetable and turkey, baby food, strained	76611010
12.5	Vegetable and turkey, baby food, junior	76611020
12.5	Puerto Rican stew (Sancocho)	77563010
25.0	Raw vegetable, NFS	75100250
25.0	Vegetables, NS as to type, cooked, NS as to fat added in coo	75200100
25.0	Vegetables, NS as to type, cooked, fat not added in cooking	75200110
25.0	Vegetable combination (including carrots, broccoli, and/or d	75440100
25.0	Vegetable combination (excluding carrots, broccoli, and dark	75440110
25.0	Vegetable tempura	75440200
25.0	Vegetables, dipped in chick-pea flour batter, (pakora), frie	75440400
25.0	Vegetable combinations (including carrots, broccoli, and/or	75440500
25.0	Vegetable combination (including carrots, broccoli, and/or d	75450500
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460700
25.0	Vegetable combinations (excluding carrots, broccoli, and dar	75460710
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460800
25.0	Vegetable soup, home recipe	75649110
25.0	Vegetable noodle soup, home recipe	75649150
25.0	Vegetable beef soup, home recipe	75652010
25.0	Vegetable beef soup with noodles or pasta, home recipe	75652040

% Protected Produce in Food Item	Food Item Description	USDA Food Code
25.0	Vegetable beef soup with rice, home recipe	75652050
25.0	Spanish stew, Puerto Rican style (Cocido Espanol)	77513010
33.0	Mixed vegetable juice (vegetables other than tomato)	75132000
33.0	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340110
33.0	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340120
33.0	Vegetable stew without meat	75439010
33.0	Mixed vegetables, garden vegetables, baby food, NS as to str	76407000

Table D.4 Food Codes for Root Vegetables

% Root Produce in Food Item	Food Item Description	USDA Food Code
33.0	Mixed vegetables, garden vegetables, baby food, strained	76407010
33.0	Mixed vegetables, garden vegetables, baby food, junior	76407020
50.0	Potato pancake	71701000
50.0	Norwegian Lefse, potato and flour pancake	71701500
50.0	Stewed potatoes, Mexican style (Papas guisadas)	71703000
50.0	Stewed potatoes with tomatoes, Mexican style (Papas guisadas	71703040
50.0	Stewed potatoes with tomatoes	71704000
50.0	Potato soup, NS as to made with milk or water	71801000
50.0	Potato soup, cream of, prepared with milk	71801010
50.0	Potato soup, prepared with water	71801020
50.0	Potato soup, instant, made from dry mix	71801040
50.0	Potato and cheese soup	71801100
50.0	Macaroni and potato soup	71802010
50.0	Potato chowder	71803010
50.0	Peas and carrots, cooked, NS as to form, NS as to fat added	73111200
50.0	Peas and carrots, cooked, from fresh, NS as to fat added in	73111201
50.0	Peas and carrots, cooked, from frozen, NS as to fat added in	73111202
50.0	Peas and carrots, cooked, NS as to form, fat not added in co	73111210
50.0	Peas and carrots, cooked, from fresh, fat not added in cooki	73111211
50.0	Peas and carrots, cooked, from frozen, fat not added in cook	73111212
50.0	Peas and carrots, cooked, NS as to form, fat added in cookin	73111220
50.0	Peas and carrots, cooked, from fresh, fat added in cooking	73111221
50.0	Peas and carrots, cooked, from frozen, fat added in cooking	73111222
50.0	Carrot soup, cream of, prepared with milk	73501000
50.0	Tomato and onion, cooked, NS as to fat added in cooking	74504100
50.0	Tomato and onion, cooked, fat not added in cooking	74504110
50.0	Tomato and onion, cooked, fat added in cooking	74504120
50.0	Beans, green, and potatoes, cooked, fat not added in cooking	75302050
50.0	Beans, green, and potatoes, cooked, NS as to fat added in co	75302500

50.0	Beans, green, and potatoes, cooked, fat added in cooking	75302510
50.0	Peas and onions, cooked, fat not added in cooking	75315110
50.0	Peas and potatoes, cooked, fat not added in cooking	75315300
50.0	Squash, summer, and onions, cooked, fat not added in cooking	75316000
50.0	Onion rings, NS as to form, batter-dipped, baked or fried	75415020
50.0	Onion rings, from fresh, batter-dipped, baked or fried	75415021
50.0	Carrots and peas, baby food, strained	76202000
50.0	Carrots and beef, baby food, strained	76602000
50.0	Sweetpotatoes and chicken, baby food, strained	76604500
75.0	White potato, cooked, with cheese	71301020
75.0	White potato, cooked, with ham and cheese	71301120
75.0	White potato, scalloped	71305010
75.0	White potato, scalloped, with ham	71305110
75.0	Carrots, cooked, from fresh, creamed	73102231
75.0	Carrots, cooked, NS as to form, glazed	73102240
75.0	Carrots, cooked, from fresh, glazed	73102241
75.0	Carrots, cooked, from frozen, glazed	73102242
75.0	Carrots, cooked, from fresh, with cheese sauce	73102251
75.0	Carrots in tomato sauce	73111400
100.0	White potato, NFS	71000100
100.0	White potato, baked, peel not eaten	71101000
100.0	White potato, baked, peel eaten, NS as to fat added in cooki	71101100
100.0	White potato, baked, peel eaten, fat not added in cooking	71101110
100.0	White potato, baked, peel eaten, fat added in cooking	71101120
100.0	White potato skins, with adhering flesh, baked	71101150
100.0	White potato, boiled, without peel, NS as to fat added in co	71103000
100.0	White potato, boiled, without peel, fat not added in cooking	71103010
100.0	White potato, boiled, without peel, fat added in cooking	71103020
100.0	White potato, boiled, with peel, NS as to fat added in cooki	71103100
100.0	White potato, boiled, with peel, fat not added in cooking	71103110
100.0	White potato, boiled, with peel, fat added in cooking	71103120
100.0	White potato, boiled, without peel, canned, low sodium, fat	71103210
100.0	White potato, roasted, NS as to fat added in cooking	71104000
100.0	White potato, roasted, fat not added in cooking	71104010
100.0	White potato, roasted, fat added in cooking	71104020

100.0	White potato, sticks	71205000
100.0	White potato skins, chips	71211000
100.0	White potato, french fries, NS as to from fresh or frozen	71401000
100.0	White potato, french fries, from fresh, deep fried	71401010
100.0	White potato, french fries, from frozen, oven baked	71401020
100.0	White potato, french fries, from frozen, deep fried	71401030
100.0	White potato, french fries, breaded or battered	71402040
100.0	White potato, home fries	71403000
100.0	White potato, home fries, with green or red peppers and onio	71403500
100.0	White potato, hash brown, NS as to from fresh, frozen, or dr	71405000
100.0	White potato, hash brown, from fresh	71405010
100.0	White potato, hash brown, from frozen	71405020
100.0	White potato, hash brown, with cheese	71405100
100.0	White potato skins, with adhering flesh, fried	71410000
100.0	White potato skins, with adhering flesh, fried, with cheese	71410500
100.0	White potato skins, with adhering flesh, fried, with cheese	71411000
100.0	White potato, mashed, NFS	71501000
100.0	White potato, from fresh, mashed, made with milk	71501010
100.0	White potato, from fresh, mashed, made with milk, sour cream	71501015
100.0	White potato, from fresh, mashed, made with milk and fat	71501020
100.0	White potato, from fresh, mashed, made with fat	71501030
100.0	White potato, from fresh, mashed, made with milk, fat and ch	71501050
100.0	White potato, from fresh, mashed, not made with milk or fat	71501080
100.0	White potato, from fresh, mashed, NS as to milk or fat	71501310
100.0	White potato, patty	71503010
100.0	White potato, puffs	71505000
100.0	White potato, stuffed, baked, peel not eaten, NS as to toppi	71507000
100.0	White potato, stuffed, baked, peel not eaten, stuffed with s	71507010
100.0	White potato, stuffed, baked, peel not eaten, stuffed with c	71507020
100.0	White potato, stuffed, baked, peel not eaten, stuffed with b	71507040
100.0	White potato, stuffed, baked, peel eaten, stuffed with sour	71508010
100.0	White potato, stuffed, baked, peel eaten, stuffed with chees	71508020
100.0	White potato, stuffed, baked, peel eaten, stuffed with chili	71508030
100.0	White potato, stuffed, baked, peel eaten, stuffed with brocc	71508040
100.0	White potato, stuffed, baked, peel eaten, stuffed with meat	71508050

100.0	White potato, stuffed, baked, peel eaten, stuffed with bacon	71508060
100.0	White potato, stuffed, baked, peel not eaten, stuffed with b	71508070
100.0	Potato salad with egg	71601010
100.0	Potato salad, German style	71602010
100.0	Potato salad	71603010
100.0	Carrots, raw	73101010
100.0	Carrots, raw, salad	73101110
100.0	Carrots, raw, salad with apples	73101210
100.0	Carrots, cooked, NS as to form, NS as to fat added in cookin	73102200
100.0	Carrots, cooked, from fresh, NS as to fat added in cooking	73102201
100.0	Carrots, cooked, from frozen, NS as to fat added in cooking	73102202
100.0	Carrots, cooked, NS as to form, fat not added in cooking	73102210
100.0	Carrots, cooked, from fresh, fat not added in cooking	73102211
100.0	Carrots, cooked, from frozen, fat not added in cooking	73102212
100.0	Carrots, cooked, NS as to form, fat added in cooking	73102220
100.0	Carrots, cooked, from fresh, fat added in cooking	73102221
100.0	Carrots, cooked, from frozen, fat added in cooking	73102222
100.0	Sweetpotato, NFS	73401000
100.0	Sweetpotato, baked, peel eaten, fat not added in cooking	73402010
100.0	Sweetpotato, baked, peel eaten, fat added in cooking	73402020
100.0	Sweetpotato, baked, peel not eaten, NS as to fat added in co	73403000
100.0	Sweetpotato, baked, peel not eaten, fat not added in cooking	73403010
100.0	Sweetpotato, baked, peel not eaten, fat added in cooking	73403020
100.0	Sweetpotato, boiled, without peel, NS as to fat added in coo	73405000
100.0	Sweetpotato, boiled, without peel, fat not added in cooking	73405010
100.0	Sweetpotato, boiled, without peel, fat added in cooking	73405020
100.0	Sweetpotato, boiled, with peel, fat not added in cooking	73405110
100.0	Sweetpotato, boiled, with peel, fat added in cooking	73405120
100.0	Sweetpotato, candied	73406000
100.0	Sweetpotato, canned, NS as to syrup	73407000
100.0	Sweetpotato, canned without syrup	73407010
100.0	Sweetpotato, canned in syrup, with fat added in cooking	73407030
100.0	Sweetpotato, casserole or mashed	73409000
100.0	Sweetpotato, fried	73410110
100.0	Beets, raw	75102500

100.0	Garlic, raw	75111500
100.0	Jicama, raw	75111800
100.0	Onions, young green, raw	75117010
100.0	Onions, mature, raw	75117020
100.0	Radish, raw	75125000
100.0	Turnip, raw	75129000
100.0	Beets, cooked, NS as to form, NS as to fat added in cooking	75208000
100.0	Beets, cooked, NS as to form, fat not added in cooking	75208010
100.0	Beets, cooked, from fresh, fat not added in cooking	75208011
100.0	Beets, cooked, NS as to form, fat added in cooking	75208020
100.0	Beets, cooked, from fresh, fat added in cooking	75208021
100.0	Garlic, cooked	75217400
100.0	Onions, mature, cooked, NS as to form, NS as to fat added in	75221000
100.0	Onions, mature, cooked, from fresh, NS as to fat added in co	75221001
100.0	Onions, mature, cooked, from frozen, NS as to fat added in c	75221002
100.0	Onions, mature, cooked, NS as to form, fat not added in cook	75221010
100.0	Onions, mature, cooked, from fresh, fat not added in cooking	75221011
100.0	Onions, mature, cooked or sauteed, NS as to form, fat added	75221020
100.0	Onions, mature, cooked or sauteed, from fresh, fat added in	75221021
100.0	Onions, mature, cooked or sauteed, from frozen, fat added in	75221022
100.0	Onions, pearl, cooked, NS as to form	75221030
100.0	Onions, pearl, cooked, from fresh	75221031
100.0	Onion, young green, cooked, NS as to form, NS as to fat adde	75221040
100.0	Onions, young green, cooked, NS as to form, fat not added in	75221050
100.0	Onions, young green, cooked, from fresh, fat not added in co	75221051
100.0	Onion, young green, cooked, from fresh, fat added in cooking	75221061
100.0	Parsnips, cooked, fat not added in cooking	75222010
100.0	Parsnips, cooked, fat added in cooking	75222020
100.0	Radish, Japanese (daikon), cooked, fat added in cooking	75227110
100.0	Turnip, cooked, from fresh, NS as to fat added in cooking	75234001
100.0	Turnip, cooked, NS as to form, fat not added in cooking	75234010
100.0	Turnip, cooked, from fresh, fat not added in cooking	75234011
100.0	Turnip, cooked, from fresh, fat added in cooking	75234021
100.0	Vegetables, stew type (including potatoes, carrots, onions,	75317000

100.0	Vegetables, stew type (including potatoes, carrots, onions,	75317010
100.0	Vegetables, stew type (including potatoes, carrots, onions,	75317020
100.0	Beets with Harvard sauce	75405010
100.0	Beets, pickled	75500210
100.0	Carrots, baby food, NS as to strained or junior	76201000
100.0	Carrots, baby food, strained	76201010
100.0	Carrots, baby food, junior	76201020
100.0	Carrots, baby food, toddler	76201030
100.0	Sweetpotatoes, baby food, NS as to strained or junior	76209000
100.0	Sweetpotatoes, baby food, strained	76209010

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
12.5	Meat loaf, NS as to type of meat	27260010
12.5	Meatballs, with breading, NS as to type of meat, with gravy	27260050
12.5	Gumbo, no rice (New Orleans type with shellfish, pork, and/o	27464000
12.5	Meat loaf dinner, NFS (frozen meal)	28160300
12.5	Meat loaf with potatoes, vegetable (frozen meal)	28160310
12.5	Meatball soup, Mexican style (Sopa de Albondigas)	28310230
12.5	Chicken soup with noodles and potatoes, Puerto Rican style	28340220
12.5	Chicken gumbo soup	28340310
12.5	Chicken noodle soup, chunky style	28340510
12.5	Chicken soup, canned, undiluted	28340520
12.5	Chicken soup	28340530
12.5	Sweet and sour soup	28340550
12.5	Chicken soup with vegetables (broccoli, carrots, celery, pot	28340580
12.5	Chicken corn soup with noodles, home recipe	28340590
12.5	Chicken or turkey vegetable soup, stew type	28340610
12.5	Chicken vegetable soup with rice, stew type, chunky style	28340630
12.5	Chicken vegetable soup with noodles, stew type, chunky style	28340640
12.5	Chicken or turkey vegetable soup, home recipe	28340660
12.5	Chicken vegetable soup with rice, Mexican style (Sopa / Cald	28340670
12.5	Hot and sour soup	28340750
12.5	Chicken soup with vegetables and fruit, Oriental Style	28340800
12.5	Chicken or turkey soup, cream of, canned, reduced sodium, ma	28345030
12.5	Chicken or turkey soup, cream of, canned, reduced sodium, un	28345040
12.5	Chicken or turkey soup, cream of, NS as to prepared with mil	28345110
12.5	Chicken or turkey soup, cream of, prepared with milk	28345120
12.5	TAMALE W/ MEAT &/OR POULTRY (INCL TAMALE, NFS)	58103110
12.5	Tamale casserole with meat	58103310
12.5	Quesadilla with meat and cheese	58104730
12.5	TAQUITOES	58104810
12.5	Meat turnover, Puerto Rican style (Pastelillo de carne; Empa	58116110

% Poultry in Food Item	Food Item Description	USDA Food Code
12.5	Empanada, Mexican turnover, filled with meat and vegetables	58116120
12.5	Dumpling, meat-filled	58121510
12.5	Quiche with meat, poultry or fish	58125110
12.5	Turnover, meat-filled, no gravy	58126110
12.5	Turnover, meat- and cheese-filled, no gravy	58126130
12.5	Turnover, meat- and bean-filled, no gravy	58126140
12.5	Turnover, meat- and cheese-filled, tomato-based sauce	58126150
12.5	Turnover, meat-and vegetable- filled (no potatoes, no gravy)	58126170
12.5	Dressing with chicken or turkey and vegetables	58128220
12.5	Stuffed pepper, with meat	58162090
12.5	Stuffed pepper, with rice and meat	58162110
12.5	Chicken noodle soup	58403010
12.5	Chicken noodle soup, home recipe	58403040
12.5	Chicken rice soup	58404010
12.5	Chicken soup with dumplings	58404520
12.5	Turkey noodle soup, home recipe	58406020
25.0	Turnover, chicken- or turkey-, and cheese-filled, no gravy	58126270
25.0	Turnover, chicken- or turkey-, and vegetable-filled, lower i	58126280
33.0	Chicken or turkey, potatoes, and vegetables (including carro	27341010
33.0	Chicken or turkey, potatoes, and vegetables (excluding carro	27341020
33.0	Chicken or turkey stew with potatoes and vegetables (includi	27341310
33.0	Chicken or turkey stew with potatoes and vegetables (excludi	27341320
33.0	Chicken or turkey stew with potatoes and vegetables (includi	27341510
33.0	Chicken or turkey stew with potatoes and vegetables (excludi	27341520
33.0	Chicken or turkey, noodles, and vegetables (including carrot	27343010
33.0	Chicken or turkey, noodles, and vegetables (excluding carrot	27343020
33.0	Chicken or turkey, noodles, and vegetables (including carrot	27343470
33.0	Chicken or turkey, noodles, and vegetables (excluding carrot	27343480
33.0	Chicken or turkey, noodles, and vegetables (including carrot	27343510
33.0	Chicken or turkey, noodles, and vegetables (excluding carrot	27343520
33.0	Chicken or turkey chow mein or chop suey with noodles	27343910
33.0	Chicken or turkey, noodles, and vegetables (including carrot	27343950

% Poultry in Food Item	Food Item Description	USDA Food Code
33.0	Chicken or turkey, noodles, and vegetables (excluding carrot	27343960
33.0	CHICKEN, NOODLES, VEG (NO CAR/DK GRN), CREAM SAUCE	27343980
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345010
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345020
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345210
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345220
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345310
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345320
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345410
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345420
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345440
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345520
33.0	Chicken or turkey pot pie	27347100
33.0	Chicken or turkey, dumplings, and vegetables (including carr	27347240
33.0	Chicken or turkey, dumplings, and vegetables (excluding carr	27347250
33.0	Chicken, fried, with potatoes, vegetable (frozen meal)	28140710
33.0	Chicken patty, or nuggets, boneless, breaded, potatoes, vege	28140720
33.0	Chicken patty, breaded, with tomato sauce and cheese, fettuc	28140730
33.0	Chicken patty, or nuggets, boneless, breaded, with pasta and	28140740
33.0	Chicken, fried, with potatoes, vegetable, dessert (frozen me	28140810
33.0	Chicken, fried, with potatoes, vegetable, dessert (frozen me	28141010
33.0	CHICKEN PATTY W/ VEGETABLES (DIET FROZEN MEAL)	28141060
33.0	CHICKEN TERIYAKI W/ RICE, VEGETABLE (FROZEN MEAL)	28141200
33.0	Chicken with rice-vegetable mixture (diet frozen meal)	28141250
33.0	Chicken with rice and vegetable, reduced fat and sodium (die	28141300
33.0	Chicken a la king with rice (frozen meal)	28141600
33.0	Chicken and vegetables in cream or white sauce (diet frozen	28141610
33.0	Chicken and vegetable entree with rice, Oriental (diet froze	28143020
33.0	Chicken and vegetable entree, oriental (diet frozen meal)	28143030
33.0	Chicken chow mein with rice (diet frozen meal)	28143040
33.0	Chicken with noodles and cheese sauce (diet frozen meal)	28143080
33.0	Chicken cacciatore with noodles (diet frozen meal)	28143110

% Poultry in Food Item	Food Item Description	USDA Food Code
33.0	Chicken and vegetable entree with noodles (frozen meal)	28143130
33.0	Chicken and vegetable entree with noodles (diet frozen meal)	28143150
33.0	Chicken in cream sauce with noodles and vegetable (frozen me	28143170
33.0	Chicken in butter sauce with potatoes and vegetable (diet fr	28143180
33.0	Chicken in soy-based sauce, rice and vegetables (frozen meal	28143200
33.0	Chicken in orange sauce with almond rice (diet frozen meal)	28143210
33.0	Chicken in barbecue sauce, with rice, vegetable and dessert,	28143220
33.0	Chicken and vegetable entree with noodles and cream sauce (f	28144100
33.0	Turkey dinner, NFS (frozen meal)	28145000
33.0	TURKEY W/ DRESSING, GRAVY, POTATO (FROZEN MEAL)	28145010
33.0	Turkey with dressing, gravy, vegetable and fruit (diet froze	28145100
33.0	Turkey with vegetable, stuffing (diet frozen meal)	28145110
33.0	Turkey with gravy, dressing, potatoes, vegetable (frozen mea	28145210
33.0	Turkey with gravy, dressing, potatoes, vegetable, dessert (f	28145610
33.0	Burrito with chicken, no beans	58100200
33.0	Burrito with chicken and beans	58100210
33.0	Burrito with chicken, beans, and cheese	58100220
33.0	Burrito with chicken and cheese	58100230
33.0	Burrito with chicken, NFS	58100240
33.0	Enchilada with chicken, tomato-based sauce	58100600
33.0	Enchilada with chicken, beans, and cheese, tomato- based sau	58100620
33.0	Enchilada with chicken and cheese, no beans, tomato- based s	58100630
33.0	Flauta with chicken	58101240
33.0	Soft taco with chicken, cheese, and lettuce	58101450
33.0	Soft taco with chicken, cheese, lettuce, tomato and sour cre	58101460
33.0	Taco or tostada with chicken or turkey, lettuce, tomato and	58101510
33.0	Taco or tostada with chicken, cheese, lettuce, tomato and sa	58101520
33.0	Nachos with chicken or turkey and cheese	58104250
33.0	Chimichanga with chicken and cheese	58104530
33.0	Fajita with chicken and vegetables	58105000
33.0	Cornmeal dressing with chicken or turkey and vegetables	58128120
33.0	Rice with chicken, Puerto Rican style (Arroz con Pollo)	58155110

% Poultry in Food Item	Food Item Description	USDA Food Code
50.0	Chicken or turkey and potatoes with gravy (mixture)	27241010
50.0	Chicken or turkey and noodles, no sauce (mixture)	27242000
50.0	Chicken or turkey and noodles with gravy (mixture)	27242200
50.0	Chicken or turkey and noodles with (mushroom) soup (mixture)	27242250
50.0	Chicken or turkey and noodles with cream or white sauce (mix	27242300
50.0	Chicken or turkey and noodles with cheese sauce (mixture)	27242310
50.0	Chicken or turkey and noodles, tomato-based sauce (mixture)	27242400
50.0	Chicken or turkey and rice, no sauce (mixture)	27243000
50.0	Chicken or turkey and rice with cream sauce (mixture)	27243300
50.0	Chicken or turkey and rice with (mushroom) soup (mixture)	27243400
50.0	Chicken or turkey and rice with tomato-based sauce (mixture)	27243500
50.0	Chicken or turkey and rice with soy-based sauce (mixture)	27243600
50.0	Chicken or turkey with dumplings (mixture)	27246100
50.0	Chicken or turkey with stuffing (mixture)	27246200
50.0	Chicken or turkey and vegetables (including carrots, broccol	27440110
50.0	Chicken or turkey and vegetables (excluding carrots, broccol	27440120
50.0	Chicken or turkey and vegetables (including carrots, broccol	27442110
50.0	Chicken or turkey and vegetables (excluding carrots, broccol	27442120
50.0	Chicken or turkey a la king with vegetables (including carro	27443110
50.0	Chicken or turkey a la king with vegetables (excluding carro	27443120
50.0	Chicken or turkey divan	27443150
50.0	Chicken or turkey and vegetables (including carrots, broccol	27445110
50.0	Chicken or turkey and vegetables (excluding carrots, broccol	27445120
50.0	General Tso (General Gau) chicken	27445150
50.0	Moo Goo Gai Pan	27445180
50.0	Kung pao chicken	27445220
50.0	Almond chicken	27445250
50.0	Chicken or turkey chow mein or chop suey, no noodles	27446100
50.0	Chicken or turkey salad	27446200
50.0	Chicken or turkey salad with egg	27446220
50.0	Chicken or turkey garden salad (chicken and/or turkey, tomat	27446300
50.0	Chicken or turkey garden salad (chicken and/or turkey, other	27446310

% Poultry in Food Item	Food Item Description	USDA Food Code
50.0	Chicken or turkey and vegetables (including carrots, broccol	27446400
75.0	Meat loaf made with chicken or turkey	27246500
75.0	Chicken sandwich, with spread	27540110
75.0	Chicken barbecue sandwich	27540130
75.0	Chicken fillet (breaded, fried) sandwich	27540140
75.0	Chicken fillet (breaded, fried) sandwich with lettuce, tomat	27540150
75.0	Chicken patty sandwich, miniature, with spread	27540170
75.0	Chicken patty sandwich or biscuit	27540180
75.0	Chicken patty sandwich, with lettuce and spread	27540190
75.0	Fajita-style chicken sandwich with cheese, on pita bread, wi	27540200
75.0	Chicken patty sandwich with cheese, on wheat bun, with lettu	27540230
75.0	Chicken fillet, (broiled), sandwich, on whole wheat roll, wi	27540240
75.0	Chicken fillet, broiled, sandwich with cheese, on whole whea	27540250
75.0	Chicken fillet, broiled, sandwich, on oat bran bun, with let	27540260
75.0	Chicken fillet, broiled, sandwich, with lettuce, tomato, and	27540270
75.0	Chicken fillet, broiled, sandwich with cheese, on bun, with	27540280
100.0	Chicken, NS as to part and cooking method, NS as to skin eat	24100000
100.0	Chicken, NS as to part and cooking method, skin eaten	24100010
100.0	Chicken, NS as to part and cooking method, skin not eaten	24100020
100.0	CHICKEN, BONELESS, BROILED, NS PART, NS SKIN	24101000
100.0	CHICKEN, BONELESS, BROILED, NS PART, W/O SKIN	24101020
100.0	Chicken, NS as to part, roasted, broiled, or baked, NS as to	24102000
100.0	Chicken, NS as to part, roasted, broiled, or baked, skin eat	24102010
100.0	Chicken, NS as to part, roasted, broiled, or baked, skin not	24102020
100.0	Chicken, NS as to part, stewed, NS as to skin eaten	24103000
100.0	Chicken, NS as to part, stewed, skin eaten	24103010
100.0	Chicken, NS as to part, stewed, skin not eaten	24103020
100.0	Chicken, NS as to part, fried, no coating, NS as to skin eat	24104000
100.0	Chicken, NS as to part, fried, no coating, skin not eaten	24104020
100.0	CHICKEN, BONELESS, FLOURED, BAKED/FRIED, NS SKIN	24105000
100.0	CHICKEN, BONELESS, FLOURED, BAKED/FRIED, W/ SKIN	24105010
100.0	CHICKEN, BONELESS, BREADED, BAKED/FRIED, NS SKIN	24106000

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	CHICKEN, BONELESS, BREADED, BAKED/FRIED, W/ SKIN	24106010
100.0	CHICKEN,BONELESS,BREADD,BAKD/FRIED,W/O SKIN,NS COAT	24106040
100.0	CHICKEN,BONELESS,BREADD,BAKED/FRIED,W/O SKIN,W/COAT	24106050
100.0	Chicken, NS as to part, coated, baked or fried, prepared wit	24107000
100.0	Chicken, NS as to part, coated, baked or fried, prepared wit	24107010
100.0	Chicken, NS as to part, coated, baked or fried, prepared wit	24107020
100.0	Chicken, NS as to part, coated, baked or fried, prepared ski	24107050
100.0	CHICKEN, W/ BONE, NFS	24110000
100.0	CHICKEN, W/ BONE, NS AS TO PART, ROASTED, W/ SKIN	24112010
100.0	CHICKEN,W/BONE,NS PART,BREADED,BAKD/FRIED, W/O SKIN	24116020
100.0	Chicken, breast, NS as to cooking method, NS as to skin eate	24120100
100.0	Chicken, breast, NS as to cooking method, skin eaten	24120110
100.0	Chicken, breast, NS as to cooking method, skin not eaten	24120120
100.0	CHICKEN, BREAST, BROILED, NS AS TO SKIN	24121100
100.0	CHICKEN, BREAST, BROILED, W/SKIN	24121110
100.0	CHICKEN, BREAST, BROILED, W/O SKIN	24121120
100.0	Chicken, breast, roasted, broiled, or baked, NS as to skin e	24122100
100.0	Chicken, breast, roasted, broiled, or baked, skin eaten	24122110
100.0	Chicken, breast, roasted, broiled, or baked, skin not eaten	24122120
100.0	Chicken, breast, stewed, NS as to skin eaten	24123100
100.0	Chicken, breast, stewed, skin eaten	24123110
100.0	Chicken, breast, stewed, skin not eaten	24123120
100.0	Chicken, breast, fried, no coating, NS as to skin eaten	24124100
100.0	Chicken, breast, fried, no coating, skin eaten	24124110
100.0	Chicken, breast, fried, no coating, skin not eaten	24124120
100.0	CHICKEN, BREAST, FLOURED,BAKED/FRIED, NS AS TO SKIN	24125100
100.0	CHICKEN, BREAST, FLOURED, BAKED/FRIED, W/ SKIN	24125110
100.0	CHICKEN, BREAST, FLOURED, BAKED/FRIED, W/O SKIN	24125120
100.0	CHICKEN,BREAST,FLOURED,BAKED/FRIED,W/O SKIN,NS COAT	24125140
100.0	CHICKEN, BREAST, BREADED,BAKED/FRIED, NS AS TO SKIN	24126100
100.0	CHICKEN, BREAST, BREADED, BAKED/FRIED, W/ SKIN	24126110
100.0	CHICKEN, BREAST, BREADED, BAKED/FRIED, W/O SKIN	24126120

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	CHICKEN,BREAST,BREADED,BAKED/FRIED, SKINLESS,W/COAT	24126150
100.0	CHICKEN,BREAST,BREADED,BAKED/FRIED,W/O SKIN,NO COAT	24126160
100.0	Chicken, breast, coated, baked or fried, prepared with skin,	24127100
100.0	Chicken, breast, coated, baked or fried, prepared with skin,	24127110
100.0	Chicken, breast, coated, baked or fried, prepared with skin,	24127120
100.0	Chicken, breast, coated, baked or fried, prepared skinless,	24127140
100.0	Chicken, breast, coated, baked or fried, prepared skinless,	24127150
100.0	Chicken, breast, coated, baked or fried, prepared skinless,	24127160
100.0	Chicken, leg (drumstick and thigh), NS as to cooking method,	24130200
100.0	Chicken, leg (drumstick and thigh), NS as to cooking method,	24130220
100.0	CHICKEN, LEG, BROILED, NS AS TO SKIN	24131200
100.0	CHICKEN, LEG, BROILED, W/ SKIN	24131210
100.0	CHICKEN, LEG, BROILED, W/O SKIN	24131220
100.0	Chicken, leg (drumstick and thigh), roasted, broiled, or bak	24132200
100.0	Chicken, leg (drumstick and thigh), roasted, broiled, or bak	24132210
100.0	Chicken, leg (drumstick and thigh), roasted, broiled, or bak	24132220
100.0	Chicken, leg (drumstick and thigh), stewed, NS as to skin ea	24133200
100.0	Chicken, leg (drumstick and thigh), stewed, skin eaten	24133210
100.0	Chicken, leg (drumstick and thigh), stewed, skin not eaten	24133220
100.0	Chicken, leg (drumstick and thigh), fried, no coating, NS as	24134200
100.0	Chicken, leg (drumstick and thigh), fried, no coating, skin	24134210
100.0	Chicken, leg (drumstick and thigh), fried, no coating, skin	24134220
100.0	CHICKEN, LEG, FLOURED, BAKED/FRIED, NS AS TO SKIN	24135200
100.0	CHICKEN, LEG, FLOURED, BAKED/FRIED, W/ SKIN	24135210
100.0	CHICKEN, LEG, FLOURED, BAKED/FRIED, W/O SKIN	24135220
100.0	CHICKEN, LEG, BREADED, BAKED/FRIED, W/ SKIN	24136210
100.0	Chicken, leg (drumstick and thigh), coated, baked or fried,	24137210
100.0	Chicken, leg (drumstick and thigh), coated, baked or fried,	24137220
100.0	Chicken, leg (drumstick and thigh), coated, baked or fried,	24137240
100.0	Chicken, leg (drumstick and thigh), coated, baked or fried,	24137250
100.0	Chicken, drumstick, NS as to cooking method, NS as to skin e	24140200
100.0	Chicken, drumstick, NS as to cooking method, skin eaten	24140210

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	Chicken, drumstick, NS as to cooking method, skin not eaten	24140220
100.0	CHICKEN, DRUMSTICK, BROILED, NS AS TO SKIN	24141200
100.0	CHICKEN, DRUMSTICK, BROILED, W/ SKIN	24141210
100.0	CHICKEN, DRUMSTICK, BROILED, W/O SKIN	24141220
100.0	Chicken, drumstick, roasted, broiled, or baked, NS as to ski	24142200
100.0	Chicken, drumstick, roasted, broiled, or baked, skin eaten	24142210
100.0	Chicken, drumstick, roasted, broiled, or baked, skin not eat	24142220
100.0	Chicken, drumstick, stewed, NS as to skin eaten	24143200
100.0	Chicken, drumstick, stewed, skin eaten	24143210
100.0	Chicken, drumstick, stewed, skin not eaten	24143220
100.0	Chicken, drumstick, fried, no coating, NS as to skin eaten	24144200
100.0	Chicken, drumstick, fried, no coating, skin eaten	24144210
100.0	Chicken, drumstick, fried, no coating, skin not eaten	24144220
100.0	CHICKEN, DRUMSTICK,FLOURED,BAKD/FRIED,NS AS TO SKIN	24145200
100.0	CHICKEN, DRUMSTICK, FLOURED, BAKED/FRIED, W/ SKIN	24145210
100.0	CHICKEN, DRUMSTICK, FLOURED, BAKED/FRIED, W/O SKIN	24145220
100.0	CHICKEN,DRUMSTICK,FLOURD,BAKD/FRID,W/O SKIN,W/ COAT	24145250
100.0	CHICKEN, DRUMSTICK, BREADED, BAKED/FRIED, W/ SKIN	24146210
100.0	CHICKEN, DRUMSTICK, BREADED, BAKED/FRIED, W/O SKIN	24146220
100.0	CHICKEN,DRUMSTICK,BREADED,BAKD/FRID,SKINLESS,W/COAT	24146250
100.0	CHICKEN,DRUMSTICK,BREADD,BAKD/FRID,W/O SKIN,NO COAT	24146260
100.0	Chicken, drumstick, coated, baked or fried, prepared with sk	24147200
100.0	Chicken, drumstick, coated, baked or fried, prepared with sk	24147210
100.0	Chicken, drumstick, coated, baked or fried, prepared with sk	24147220
100.0	Chicken, drumstick, coated, baked or fried, prepared skinles	24147240
100.0	Chicken, drumstick, coated, baked or fried, prepared skinles	24147250
100.0	Chicken, drumstick, coated, baked or fried, prepared skinles	24147260
100.0	Chicken, thigh, NS as to cooking method, NS as to skin eaten	24150200
100.0	Chicken, thigh, NS as to cooking method, skin eaten	24150210
100.0	Chicken, thigh, NS as to cooking method, skin not eaten	24150220
100.0	CHICKEN, THIGH, BROILED, NS AS TO SKIN	24151200
100.0	CHICKEN, THIGH, BROILED, W/ SKIN	24151210

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	CHICKEN, THIGH, BROILED, W/O SKIN	24151220
100.0	Chicken, thigh, roasted, broiled, or baked, NS as to skin e	24152200
100.0	Chicken, thigh, roasted, broiled, or baked, skin eaten	24152210
100.0	Chicken, thigh, roasted, broiled, or baked, skin not eaten	24152220
100.0	Chicken, thigh, stewed, NS as to skin eaten	24153200
100.0	Chicken, thigh, stewed, skin eaten	24153210
100.0	Chicken, thigh, stewed, skin not eaten	24153220
100.0	Chicken, thigh, fried, no coating, NS as to skin eaten	24154200
100.0	Chicken, thigh, fried, no coating, skin eaten	24154210
100.0	Chicken, thigh, fried, no coating, skin not eaten	24154220
100.0	CHICKEN, THIGH, FLOURED, BAKED/FRIED, NS AS TO SKIN	24155200
100.0	CHICKEN, THIGH, FLOURED, BAKED/FRIED, W/ SKIN	24155210
100.0	CHICKEN, THIGH, FLOURED, BAKED/FRIED, W/O SKIN	24155220
100.0	CHICKEN, THIGH, BREADED, BAKED/FRIED, W/ SKIN	24156210
100.0	CHICKEN, THIGH, BREADED, BAKED/FRIED, W/O SKIN	24156220
100.0	CHICKEN,THIGH,BREADED,BAKD/FRIED,SKINLESS,W/COATING	24156250
100.0	CHICKEN,THIGH,BREADED,BAKED/FRIED,W/O SKIN,NO COAT	24156260
100.0	Chicken, thigh, coated, baked or fried, prepared with skin,	24157200
100.0	Chicken, thigh, coated, baked or fried, prepared with skin,	24157210
100.0	Chicken, thigh, coated, baked or fried, prepared with skin,	24157220
100.0	Chicken, thigh, coated, baked or fried, prepared skinless, N	24157240
100.0	Chicken, thigh, coated, baked or fried, prepared skinless, c	24157250
100.0	Chicken, thigh, coated, baked or fried, prepared skinless, c	24157260
100.0	Chicken, wing, NS as to cooking method, NS as to skin eaten	24160100
100.0	Chicken, wing, NS as to cooking method, skin eaten	24160110
100.0	Chicken, wing, NS as to cooking method, skin not eaten	24160120
100.0	CHICKEN, WING, BROILED, W/ SKIN	24161110
100.0	CHICKEN, WING, BROILED, W/O SKIN	24161120
100.0	Chicken, wing, roasted, broiled, or baked, NS as to skin eat	24162100
100.0	Chicken, wing, roasted, broiled, or baked, skin eaten	24162110
100.0	Chicken, wing, roasted, broiled, or baked, skin not eaten	24162120
100.0	Chicken, wing, stewed, NS as to skin eaten	24163100

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	Chicken, wing, stewed, skin eaten	24163110
100.0	Chicken, wing, stewed, skin not eaten	24163120
100.0	Chicken, wing, fried, no coating, NS as to skin eaten	24164100
100.0	Chicken, wing, fried, no coating, skin eaten	24164110
100.0	Chicken, wing, fried, no coating, skin not eaten	24164120
100.0	CHICKEN, WING, FLOURED, BAKED/FRIED, NS AS TO SKIN	24165100
100.0	CHICKEN, WING, FLOURED, BAKED/FRIED, W/ SKIN	24165110
100.0	CHICKEN, WING, FLOURED, BAKED/FRIED, W/O SKIN	24165120
100.0	CHICKEN, WING, BREADED, BAKED/FRIED, W/ SKIN	24166110
100.0	CHICKEN, WING, BREADED, BAKED/FRIED, W/O SKIN	24166120
100.0	Chicken, wing, coated, baked or fried, prepared with skin, N	24167100
100.0	Chicken, wing, coated, baked or fried, prepared with skin, s	24167110
100.0	Chicken, wing, coated, baked or fried, prepared with skin, s	24167120
100.0	Chicken, back	24170200
100.0	CHICKEN, BACK, ROASTED, W/O SKIN	24172220
100.0	CHICKEN, BACK, STEWED, NS AS TO SKIN	24173200
100.0	CHICKEN, BACK, STEWED, W/ SKIN	24173210
100.0	Chicken, neck or ribs	24180200
100.0	Chicken skin	24198440
100.0	Chicken feet	24198500
100.0	CHICKEN, CANNED, MEAT ONLY, LIGHT MEAT	24198550
100.0	Chicken, canned, meat only	24198570
100.0	CHICKEN ROLL, ROASTED, NS AS TO LIGHT OR DARK MEAT	24198640
100.0	Chicken patty, fillet, or tenders, breaded, cooked	24198700
100.0	Chicken, ground	24198720
100.0	Chicken nuggets	24198740
100.0	Chicken crackling, Puerto Rican style (Chicharron de pollo)	24198840
100.0	Turkey, NFS	24201000
100.0	Turkey, light meat, cooked, NS as to skin eaten	24201010
100.0	Turkey, light meat, cooked, skin not eaten	24201020
100.0	Turkey, light meat, cooked, skin eaten	24201030
100.0	Turkey, light meat, breaded, baked or fried, NS as to skin e	24201050

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	Turkey, light meat, breaded, baked or fried, skin not eaten	24201060
100.0	Turkey, light meat, roasted, NS as to skin eaten	24201110
100.0	Turkey, light meat, roasted, skin not eaten	24201120
100.0	Turkey, light meat, roasted, skin eaten	24201130
100.0	Turkey, dark meat, roasted, NS as to skin eaten	24201210
100.0	Turkey, dark meat, roasted, skin not eaten	24201220
100.0	Turkey, light and dark meat, roasted, NS as to skin eaten	24201310
100.0	Turkey, light and dark meat, roasted, skin not eaten	24201320
100.0	Turkey, light and dark meat, roasted, skin eaten	24201330
100.0	Turkey, light or dark meat, battered, fried, skin not eaten	24201360
100.0	Turkey, light or dark meat, stewed, NS as to skin eaten	24201400
100.0	Turkey, light or dark meat, stewed, skin not eaten	24201410
100.0	Turkey, light or dark meat, smoked, cooked, NS as to skin ea	24201500
100.0	Turkey, light or dark meat, smoked, cooked, skin not eaten	24201520
100.0	Turkey, drumstick, cooked, skin not eaten	24202010
100.0	Turkey, drumstick, cooked, skin eaten	24202020
100.0	Turkey, drumstick, roasted, NS as to skin eaten	24202050
100.0	Turkey, drumstick, roasted, skin not eaten	24202060
100.0	Turkey, drumstick, roasted, skin eaten	24202070
100.0	Turkey, thigh, cooked, NS as to skin eaten	24202450
100.0	Turkey, thigh, cooked, skin eaten	24202460
100.0	Turkey, thigh, cooked, skin not eaten	24202500
100.0	Turkey, neck, cooked	24202600
100.0	Turkey, wing, cooked, NS as to skin eaten	24203000
100.0	Turkey, wing, cooked, skin not eaten	24203010
100.0	Turkey, wing, cooked, skin eaten	24203020
100.0	Turkey, rolled roast, light or dark meat, cooked	24204000
100.0	Turkey, canned	24206000
100.0	Turkey, ground	24207000
100.0	Turkey, nuggets	24208000
100.0	CHICKEN LIVER, BATTERED, FRIED	25110410
100.0	Chicken liver, braised	25110420

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	CHICKEN LIVER, FRIED OR SAUTEED, NO COATING	25110440
100.0	Chicken liver, fried	25110450
100.0	Liver paste or pate, chicken	25112200
100.0	Chicken or turkey cake, patty, or croquette	27246300

Table D. 6 Food Codes for Beef Items

% Beef in Food Item	Food Item Description	USDA food code value
100.0	Beef, NS as to cut, cooked, NS as to fat eaten	21000100
100.0	Beef, NS as to cut, cooked, lean and fat eaten	21000110
100.0	Beef, NS as to cut, cooked, lean only eaten	21000120
100.0	Steak, NS as to type of meat, cooked, NS as to fat eaten	21001000
100.0	Steak, NS as to type of meat, cooked, lean and fat eaten	21001010
100.0	Steak, NS as to type of meat, cooked, lean only eaten	21001020
100.0	Beef, pickled	21002000
100.0	Beef, NS as to cut, fried, NS to fat eaten	21003000
100.0	Beef steak, NS as to cooking method, NS as to fat eaten	21101000
100.0	Beef steak, NS as to cooking method, lean and fat eaten	21101010
100.0	Beef steak, NS as to cooking method, lean only eaten	21101020
100.0	Beef steak, broiled or baked, NS as to fat eaten	21101110
100.0	Beef steak, broiled or baked, lean and fat eaten	21101120
100.0	Beef steak, broiled or baked, lean only eaten	21101130
100.0	Beef steak, fried, NS as to fat eaten	21102110
100.0	Beef steak, fried, lean and fat eaten	21102120
100.0	Beef steak, fried, lean only eaten	21102130
100.0	Beef steak, breaded or floured, baked or fried, NS as to fat	21103110
100.0	Beef steak, breaded or floured, baked or fried, lean and fat	21103120
100.0	Beef steak, breaded or floured, baked or fried, lean only ea	21103130
100.0	Beef steak, battered, fried, NS as to fat eaten	21104110
100.0	Beef steak, battered, fried, lean and fat eaten	21104120
100.0	Beef steak, battered, fried, lean only eaten	21104130
100.0	Beef steak, braised, NS as to fat eaten	21105110
100.0	Beef steak, braised, lean and fat eaten	21105120
100.0	Beef steak, braised, lean only eaten	21105130
100.0	Beef, oxtails, cooked	21301000
100.0	Beef, neck bones, cooked	21302000
100.0	Beef, shortribs, cooked, NS as to fat eaten	21304000
100.0	Beef, shortribs, cooked, lean and fat eaten	21304110

% Beef in Food Item	Food Item Description	USDA food code value
100.0	Beef, shortribs, cooked, lean only eaten	21304120
100.0	Beef, shortribs, barbecued, with sauce, NS as to fat eaten	21304200
100.0	Beef, shortribs, barbecued, with sauce, lean and fat eaten	21304210
100.0	Beef, shortribs, barbecued, with sauce, lean only eaten	21304220
100.0	Beef, cow head, cooked	21305000
100.0	Beef, roast, roasted, NS as to fat eaten	21401000
100.0	Beef, roast, roasted, lean and fat eaten	21401110
100.0	Beef, roast, roasted, lean only eaten	21401120
100.0	Beef, roast, canned	21401400
100.0	Beef, pot roast, braised or boiled, NS as to fat eaten	21407000
100.0	Beef, pot roast, braised or boiled, lean and fat eaten	21407110
100.0	Beef, pot roast, braised or boiled, lean only eaten	21407120
100.0	Beef, stew meat, cooked, NS as to fat eaten	21410000
100.0	Beef, stew meat, cooked, lean and fat eaten	21410110
100.0	Beef, stew meat, cooked, lean only eaten	21410120
100.0	Beef brisket, cooked, NS as to fat eaten	21417100
100.0	Beef brisket, cooked, lean and fat eaten	21417110
100.0	Beef brisket, cooked, lean only eaten	21417120
100.0	Beef, sandwich steak (flaked, formed, thinly sliced)	21420100
100.0	Ground beef or patty, cooked, NS as to regular, lean, or ext	21500100
100.0	Ground beef, meatballs, meat only, cooked, NS as to regular,	21500110
100.0	Ground beef or patty, breaded, cooked	21500200
100.0	Ground beef, regular, cooked	21501000
100.0	Ground beef, lean, cooked	21501200
100.0	Ground beef, extra lean, cooked	21501300
100.0	Beef, bacon, cooked	21601000
100.0	Beef, bacon, cooked, lean only eaten	21601250
100.0	Beef, dried, chipped, uncooked	21602000
100.0	Beef jerky	21602100
100.0	Beef, pastrami (beef, smoked, spiced)	21603000
100.0	Beef, baby food, strained	21701010
100.0	Beef liver, braised	25110120

% Beef in Food Item	Food Item Description	USDA food code value
100.0	Beef liver, fried	25110140
100.0	Beef sausage, NFS	25220100
100.0	Beef sausage, fresh, bulk, patty or link, cooked	25220140
66.0	Beef with tomato-based sauce (mixture)	27111000
66.0	Spaghetti sauce with beef or meat other than lamb or mutton,	27111050
66.0	Beef goulash	27111100
66.0	Mexican style beef stew, no potatoes, tomato-based sauce (mi	27111300
66.0	Mexican style beef stew, no potatoes, with chili peppers, to	27111310
66.0	Beef sloppy joe (no bun)	27111500
66.0	Beef with gravy (mixture)	27112000
66.0	Salisbury steak with gravy (mixture)	27112010
66.0	Beef stroganoff	27113100
66.0	Creamed chipped or dried beef	27113200
66.0	Beef with (mushroom) soup (mixture)	27114000
66.0	Beef with soy-based sauce (mixture)	27115000
66.0	Steak teriyaki with sauce (mixture)	27115100
66.0	Beef with barbecue sauce (mixture)	27116200
66.0	Beef with sweet and sour sauce (mixture)	27116300
66.0	Stewed, seasoned, ground beef, Mexican style (Picadillo de c	27116350
66.0	Stewed seasoned ground beef, Puerto Rican style (Picadillo g	27118120
33.0	Beef and potatoes, no sauce (mixture)	27211000
33.0	Beef stew with potatoes, tomato-based sauce (mixture)	27211100
33.0	Mexican style beef stew with potatoes, tomato-based sauce (m	27211110
33.0	Beef goulash with potatoes	27211150
33.0	Beef and potatoes with cream sauce, white sauce or mushroom	27211190
33.0	Beef stew with potatoes, gravy	27211200
33.0	Beef and potatoes with cheese sauce (mixture)	27211500
33.0	Stewed, seasoned, ground beef with potatoes, Mexican style (	27211550
33.0	Beef and noodles, no sauce (mixture)	27212000
33.0	Beef and macaroni with cheese sauce (mixture)	27212050
33.0	Beef and noodles with tomato-based sauce (mixture)	27212100
33.0	Chili con carne with beans and macaroni	27212120

% Beef in Food Item	Food Item Description	USDA food code value
33.0	Beef goulash with noodles	27212150
33.0	Beef and noodles with gravy (mixture)	27212200
33.0	Beef and noodles with cream or white sauce (mixture)	27212300
33.0	Beef stroganoff with noodles	27212350
33.0	Beef and noodles with (mushroom) soup (mixture)	27212400
33.0	Beef and rice, no sauce (mixture)	27213000
33.0	Beef and rice with tomato-based sauce (mixture)	27213100
33.0	Porcupine balls with tomato-based sauce (mixture)	27213120
33.0	Chili con carne with beans and rice	27213150
33.0	Beef and rice with gravy (mixture)	27213200
33.0	Beef and rice with cream sauce (mixture)	27213300
33.0	Beef and rice with soy-based sauce (mixture)	27213500
66.0	Meat loaf made with beef	27214100
66.0	Meat loaf made with beef, with tomato-based sauce	27214110
12.5	Meat loaf, NS as to type of meat	27260010
12.5	Meatballs, with breading, NS as to type of meat, with gravy	27260050
50.0	Meat loaf made with beef and pork	27260080
33.0	Meat loaf made with beef, veal and pork	27260090
66.0	Beef, potatoes, and vegetables (including carrots, broccoli,	27311110
33.0	Beef stew with potatoes and vegetables (including carrots, b	27311310
33.0	Beef stew with potatoes and vegetables (excluding carrots, b	27311320
33.0	Beef stew with potatoes and vegetables (including carrots, b	27311410
33.0	Beef stew with potatoes and vegetables (excluding carrots, b	27311420
33.0	Shepherd's pie with beef	27311510
33.0	Beef, potatoes, and vegetables (including carrots, broccoli,	27311610
33.0	Beef, potatoes, and vegetables (excluding carrots, broccoli,	27311620
33.0	Beef, noodles, and vegetables (including carrots, broccoli,	27313010
33.0	Beef, noodles, and vegetables (excluding carrots, broccoli,	27313020
33.0	Beef chow mein or chop suey with noodles	27313110
33.0	Beef, noodles, and vegetables (including carrots, broccoli,	27313150
33.0	Beef, noodles, and vegetables (excluding carrots, broccoli,	27313160
33.0	Beef, noodles, and vegetables (including carrots, broccoli,	27313210

% Beef in Food Item	Food Item Description	USDA food code value
33.0	Beef, noodles, and vegetables (excluding carrots, broccoli,	27313220
33.0	Beef, noodles, and vegetables (including carrots, broccoli,	27313410
33.0	Beef, noodles, and vegetables (excluding carrots, broccoli,	27313420
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315010
33.0	Beef, rice, and vegetables (excluding carrots, broccoli, and	27315020
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315210
33.0	Beef, rice, and vegetables (excluding carrots, broccoli, and	27315220
33.0	Stuffed cabbage rolls with beef and rice	27315250
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315310
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315410
33.0	Beef, rice, and vegetables (excluding carrots, broccoli, and	27315420
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315510
33.0	Beef, rice, and vegetables (excluding carrots, broccoli, and	27315520
33.0	Beef pot pie	27317010
50.0	Beef and vegetables (including carrots, broccoli, and/or dar	27410210
50.0	Beef and vegetables (excluding carrots, broccoli, and dark-g	27410220
50.0	Beef shish kabob with vegetables, excluding potatoes	27410250
50.0	Beef with vegetables (including carrots, broccoli, and/or da	27411100
50.0	Swiss steak	27411120
50.0	Beef rolls, stuffed with vegetables or meat mixture, tomato	27411150
50.0	Beef with vegetables (excluding carrots, broccoli, and dark	27411200
50.0	Beef and vegetables (including carrots, broccoli, and/or dar	27415100
50.0	Beef, tofu, and vegetables (including carrots, broccoli, and	27415120
50.0	Beef chow mein or chop suey, no noodles	27415150
100.0	Pepper steak	27416150
66.0	Beef steak with onions, Puerto Rican style (mixture) (Biftec	27418410
100.0	Liver, beef or calves, and onions	27460750
66.0	Beef barbecue sandwich or Sloppy Joe, on bun	27510110
66.0	Cheeseburger, plain, on bun	27510210
66.0	Cheeseburger, with mayonnaise or salad dressing, on bun	27510220
66.0	Cheeseburger, with mayonnaise or salad dressing and tomatoes	27510230
66.0	Cheeseburger, 1/4 lb meat, plain, on bun	27510240

% Beef in Food Item	Food Item Description	USDA food code value
66.0	Cheeseburger, 1/4 lb meat, with mayonnaise or salad dressing	27510250
66.0	Cheeseburger, 1/4 lb meat, with mushrooms in sauce, on bun	27510260
66.0	Double cheeseburger (2 patties), plain, on bun	27510270
66.0	Double cheeseburger (2 patties), with mayonnaise or salad dr	27510280
66.0	Double cheeseburger (2 patties), plain, on double-decker bun	27510290
66.0	Double cheeseburger (2 patties), with mayonnaise or salad dr	27510300
66.0	Cheeseburger with tomato and/or catsup, on bun	27510310
66.0	Cheeseburger, 1 oz meat, plain, on miniature bun	27510311
66.0	Cheeseburger, 1/4 lb meat, with tomato and/or catsup, on bun	27510320
66.0	Double cheeseburger (2 patties), with tomato and/or catsup,	27510330
66.0	Double cheeseburger (2 patties), with mayonnaise or salad dr	27510340
66.0	Cheeseburger, 1/4 lb meat, with mayonnaise or salad dressing	27510350
66.0	Cheeseburger with mayonnaise or salad dressing, tomato and b	27510360
66.0	Double cheeseburger (2 patties, 1/4 lb meat each), with mayo	27510370
66.0	Triple cheeseburger (3 patties, 1/4 lb meat each), with mayo	27510380
66.0	Double bacon cheeseburger (2 patties, 1/4 lb meat each), on	27510390
66.0	Bacon cheeseburger, 1/4 lb meat, with tomato and/or catsup,	27510400
66.0	Double bacon cheeseburger (2 patties, 1/4 lb meat each), wit	27510430
66.0	Bacon cheeseburger, 1/4 lb meat, with mayonnaise or salad dr	27510440
66.0	Hamburger, plain, on bun	27510500
66.0	Hamburger, with tomato and/or catsup, on bun	27510510
66.0	Hamburger, with mayonnaise or salad dressing and tomatoes, o	27510520
66.0	Hamburger, 1/4 lb meat, plain, on bun	27510530
66.0	Double hamburger (2 patties), with tomato and/or catsup, on	27510540
66.0	Hamburger, 1/4 lb meat, with mayonnaise or salad dressing an	27510560
66.0	Hamburger, with mayonnaise or salad dressing, on bun	27510590
66.0	Hamburger, 1 oz meat, plain, on miniature bun	27510600
66.0	Hamburger, 1/4 lb meat, with tomato and/or catsup, on bun	27510620
66.0	Double hamburger (2 patties), with mayonnaise or salad dress	27510660
66.0	Double hamburger (2 patties), with mayonnaise or salad dress	27510670
66.0	Double hamburger (2 patties, 1/4 lb meat each), with tomato	27510680
66.0	Double hamburger (2 patties, 1/4 lb meat each), with mayonna	27510690

% Beef in Food Item	Food Item Description	USDA food code value
66.0	Meatball and spaghetti sauce submarine sandwich	27510700
66.0	Roast beef sandwich	27513010
66.0	Roast beef submarine sandwich, with lettuce, tomato and spre	27513040
66.0	Roast beef sandwich with cheese	27513050
66.0	Roast beef sandwich with bacon and cheese sauce	27513060
66.0	Steak submarine sandwich with lettuce and tomato	27515000
66.0	Steak sandwich, plain, on roll	27515010
50.0	Beef dinner, NFS (frozen meal)	28110000
50.0	Beef with potatoes (frozen meal, large meat portion)	28110120
50.0	Beef with vegetable (diet frozen meal)	28110150
33.0	Sirloin, chopped, with gravy, mashed potatoes, vegetable (fr	28110220
33.0	Sirloin beef with gravy, potatoes, vegetable (frozen meal)	28110270
33.0	Salisbury steak dinner, NFS (frozen meal)	28110300
33.0	Salisbury steak with gravy, potatoes, vegetable (frozen meal	28110310
33.0	Salisbury steak with gravy, whipped potatoes, vegetable, des	28110330
33.0	Salisbury steak with gravy, potatoes, vegetable, dessert (fr	28110350
33.0	Salisbury steak with gravy, macaroni and cheese, vegetable (	28110370
33.0	Salisbury steak with gravy, macaroni and cheese (frozen meal	28110380
33.0	Salisbury steak, potatoes, vegetable, dessert (diet frozen m	28110390
33.0	Beef, sliced, with gravy, potatoes, vegetable (frozen meal)	28110510
12.5	Meat loaf dinner, NFS (frozen meal)	28160300
12.5	Meat loaf with potatoes, vegetable (frozen meal)	28160310
25.0	Chili beef soup	28310210
12.5	Meatball soup, Mexican style (Sopa de Albondigas)	28310230
25.0	Beef and rice noodle soup, Oriental style (Vietnamese Pho Bo	28310330
25.0	Beef and rice soup, Puerto Rican style	28310420
25.0	Pepperpot (tripe) soup	28311010
25.0	Beef vegetable soup with potato, stew type	28315100
25.0	Beef vegetable soup with noodles, stew type, chunky style	28315120
25.0	Beef vegetable soup with rice, stew type, chunky style	28315130
25.0	Beef vegetable soup, Mexican style (Sopa / caldo de Res)	28315140
33.0	Burrito with beef, no beans	58100100

% Beef in Food Item	Food Item Description	USDA food code value
33.0	Burrito with beef and beans	58100110
33.0	Burrito with beef, beans, and cheese	58100120
33.0	Burrito with beef and cheese, no beans	58100130
33.0	Burrito with beef, beans, cheese, and sour cream	58100140
33.0	Burrito with beef and potato, no beans	58100150
33.0	Enchilada with beef, no beans	58100400
33.0	Enchilada with beef and beans	58100510
33.0	Enchilada with beef, beans, and cheese	58100520
33.0	Enchilada with beef and cheese, no beans	58100530
33.0	Flauta with beef	58101230
33.0	Taco or tostada with beef, cheese and lettuce	58101300
33.0	Taco or tostada with beef, lettuce, tomato and salsa	58101310
33.0	Taco or tostada with beef, cheese, lettuce, tomato and salsa	58101320
33.0	Soft taco with beef, cheese, lettuce, tomato and sour cream	58101350
33.0	Soft taco with beef, cheese, and lettuce	58101400
33.0	Mexican casserole made with ground beef, tomato sauce, chees	58101830
33.0	Taco or tostada salad with beef and cheese, corn chips	58101910
33.0	Taco or tostada salad with beef, beans and cheese, fried flo	58101930
12.5	Tamale casserole with meat	58103310
33.0	Nachos with beef, beans, cheese, and sour cream	58104080
33.0	Nachos with beef, beans, cheese, tomatoes, sour cream and on	58104180
33.0	Chimichanga with beef and tomato	58104450
33.0	Chimichanga, NFS	58104490
33.0	Chimichanga with beef, beans, lettuce and tomato	58104500
33.0	Chimichanga with beef, cheese, lettuce and tomato	58104510
12.5	Quesadilla with meat and cheese	58104730
33.0	Fajita with beef and vegetables	58105050
25.0	Macaroni or noodles with cheese and beef	58145130
12.5	Stuffed pepper, with meat	58162090
12.5	Stuffed pepper, with rice and meat	58162110
12.5	Barley soup	58401010
12.5	Beef noodle soup	58402010

% Beef in Food Item	Food Item Description	USDA food code value
12.5	Beef dumpling soup	58402020
12.5	Beef rice soup	58402030
12.5	Beef noodle soup, home recipe	58402100

Table D.7 Food Codes for Pork Items

% Pork in Food Item	Food Item Description	USDA food code value
12.5	Meat loaf, NS as to type of meat	27260010
12.5	Meatballs, with breading, NS as to type of meat, with gravy	27260050
12.5	Meat loaf dinner, NFS (frozen meal)	28160300
12.5	Meat loaf with potatoes, vegetable (frozen meal)	28160310
12.5	Meatball soup, Mexican style (Sopa de Albondigas)	28310230
12.5	Tamale casserole with meat	58103310
12.5	Quesadilla with meat and cheese	58104730
12.5	TAQUITOES	58104810
12.5	Stuffed pepper, with meat	58162090
12.5	Stuffed pepper, with rice and meat	58162110
25.0	Brunswick stew	27360100
25.0	Gumbo, no rice (New Orleans type with shellfish, pork, and/o	27464000
25.0	Meat and corn hominy soup, Mexican style (Pozole)	28315150
25.0	Pork and rice soup, stew type, chunky style	28320110
25.0	Pork, vegetable soup with potatoes, stew type	28320150
25.0	Pork with vegetable (excluding carrots, broccoli and/or dark	28320300
33.0	Meat loaf made with beef, veal and pork	27260090
33.0	Ham or pork, noodles, and vegetables (including carrots, bro	27320070
33.0	Pork, potatoes, and vegetables (excluding carrots, broccoli,	27320110
33.0	Pork, potatoes, and vegetables (excluding carrots, broccoli,	27320210
33.0	Pork chow mein or chop suey with noodles	27320310
33.0	Pork and vegetables (including carrots, broccoli, and/or dar	27420060
33.0	Greens with ham or pork (mixture)	27420080
33.0	Moo Shu (Mu Shi) Pork, without Chinese pancake	27420160
33.0	Pork and vegetables (excluding carrots, broccoli, and dark-g	27420350
33.0	Pork chow mein or chop suey, no noodles	27420390
33.0	Pork and vegetables (excluding carrots, broccoli, and dark	27420410
33.0	Sausage and vegetables (including carrots, broccoli, and/or	27420450
33.0	Sausage and vegetables (excluding carrots, broccoli, and dar	27420460
33.0	Sausage and peppers, no sauce (mixture)	27420470
33.0	Pork and vegetables (including carrots, broccoli, and/or dar	27420500

% Pork in Food Item	Food Item Description	USDA food code value
33.0	Pork and vegetables (excluding carrots, broccoli, and dark	27420510
33.0	Burrito with pork and beans	58100180
50.0	Meat loaf made with beef and pork	27260080
50.0	Ham or pork salad	27420020
66.0	Pork and rice with tomato-based sauce (mixture)	27220110
66.0	Sausage and rice with tomato-based sauce (mixture)	27220120
66.0	Sausage and rice with (mushroom) soup (mixture)	27220150
66.0	Sausage and noodles with cream or white sauce (mixture)	27220190
66.0	Ham or pork and rice, no sauce (mixture)	27220310
66.0	Ham or pork and potatoes with gravy (mixture)	27220510
66.0	Stewed pig's feet, Puerto Rican style (Patitas de cerdo guis	27221100
66.0	Mexican style pork stew, with potatoes, tomato-based sauce (	27221150
66.0	Pork sandwich, on white roll, with onions, dill pickles and	27520500
100.0	Pork, NS as to cut, cooked, NS as to fat eaten	22000100
100.0	Pork, NS as to cut, cooked, lean and fat eaten	22000110
100.0	Pork, NS as to cut, cooked, lean only eaten	22000120
100.0	Pork, NS as to cut, fried, NS as to fat eaten	22000200
100.0	Pork, NS as to cut, fried, lean and fat eaten	22000210
100.0	Pork, NS as to cut, fried, lean only eaten	22000220
100.0	Pork, NS as to cut, breaded or floured, fried, NS as to fat	22000300
100.0	Pork, NS as to cut, breaded or floured, fried, lean and fat	22000310
100.0	Pork, NS as to cut, breaded or floured, fried, lean only eat	22000320
100.0	Pork, pickled, NS as to cut	22001000
100.0	Pork, ground or patty, cooked	22002000
100.0	Pork, ground or patty, breaded, cooked	22002100
100.0	Pork jerky	22002800
100.0	Pork chop, NS as to cooking method, NS as to fat eaten	22101000
100.0	Pork chop, NS as to cooking method, lean and fat eaten	22101010
100.0	Pork chop, NS as to cooking method, lean only eaten	22101020
100.0	Pork chop, broiled or baked, NS as to fat eaten	22101100
100.0	Pork chop, broiled or baked, lean and fat eaten	22101110
100.0	Pork chop, broiled or baked, lean only eaten	22101120

% Pork in Food Item	Food Item Description	USDA food code value
100.0	Pork chop, breaded or floured, broiled or baked, lean and fa	22101140
100.0	Pork chop, breaded or floured, broiled or baked, lean only e	22101150
100.0	Pork chop, fried, NS as to fat eaten	22101200
100.0	Pork chop, fried, lean and fat eaten	22101210
100.0	Pork chop, fried, lean only eaten	22101220
100.0	Pork chop, breaded or floured, fried, NS as to fat eaten	22101300
100.0	Pork chop, breaded or floured, fried, lean and fat eaten	22101310
100.0	Pork chop, breaded or floured, fried, lean only eaten	22101320
100.0	Pork chop, battered, fried, NS as to fat eaten	22101400
100.0	Pork chop, battered, fried, lean and fat eaten	22101410
100.0	Pork chop, battered, fried, lean only eaten	22101420
100.0	Pork chop, stewed, NS as to fat eaten	22101500
100.0	Pork chop, stewed, lean and fat eaten	22101510
100.0	Pork chop, stewed, lean only eaten	22101520
100.0	Pork chop, smoked or cured, cooked, lean and fat eaten	22107010
100.0	Pork chop, smoked or cured, cooked, lean only eaten	22107020
100.0	Pork steak or cutlet, NS as to cooking method, NS as to fat	22201000
100.0	Pork steak or cutlet, NS as to cooking method, lean and fat	22201010
100.0	Pork steak or cutlet, NS as to cooking method, lean only eat	22201020
100.0	Pork steak or cutlet, battered, fried, NS as to fat eaten	22201050
100.0	Pork steak or cutlet, battered, fried, lean and fat eaten	22201060
100.0	Pork steak or cutlet, battered, fried, lean only eaten	22201070
100.0	Pork steak or cutlet, broiled or baked, NS as to fat eaten	22201100
100.0	Pork steak or cutlet, broiled or baked, lean and fat eaten	22201110
100.0	Pork steak or cutlet, broiled or baked, lean only eaten	22201120
100.0	Pork steak or cutlet, fried, NS as to fat eaten	22201200
100.0	Pork steak or cutlet, fried, lean and fat eaten	22201210
100.0	Pork steak or cutlet, fried, lean only eaten	22201220
100.0	Pork steak or cutlet, breaded or floured, broiled or baked,	22201310
100.0	Pork steak or cutlet, breaded or floured, broiled or baked,	22201320
100.0	Pork steak or cutlet, breaded or floured, fried, NS as to fa	22201400
100.0	Pork steak or cutlet, breaded or floured, fried, lean and fa	22201410

% Pork in Food Item	Food Item Description	USDA food code value
100.0	Pork steak or cutlet, breaded or floured, fried, lean only e	22201420
100.0	Pork, tenderloin, cooked, NS as to cooking method	22210300
100.0	Pork, tenderloin, breaded, fried	22210310
100.0	Pork, tenderloin, braised	22210350
100.0	Pork, tenderloin, baked	22210400
100.0	Pork roast, NS as to cut, cooked, NS as to fat eaten	22400100
100.0	Pork roast, NS as to cut, cooked, lean and fat eaten	22400110
100.0	Pork roast, NS as to cut, cooked, lean only eaten	22400120
100.0	Pork roast, loin, cooked, NS as to fat eaten	22401000
100.0	Pork roast, loin, cooked, lean and fat eaten	22401010
100.0	Pork roast, loin, cooked, lean only eaten	22401020
100.0	Pork roast, shoulder, cooked, lean only eaten	22411020
100.0	Pork roast, smoked or cured, cooked, NS as to fat eaten	22421000
100.0	Pork roast, smoked or cured, cooked, lean and fat eaten	22421010
100.0	Pork roast, smoked or cured, cooked, lean only eaten	22421020
100.0	Canadian bacon, cooked	22501010
100.0	Bacon, NS as to type of meat, cooked	22600100
100.0	Pork bacon, NS as to fresh, smoked or cured, cooked	22600200
100.0	Pork bacon, smoked or cured, cooked	22601000
100.0	Pork bacon, smoked or cured, cooked, lean only eaten	22601020
100.0	Bacon or side pork, fresh, cooked	22601040
100.0	Pork bacon, smoked or cured, lower sodium	22602010
100.0	Pork bacon, formed, lean meat added, cooked	22605010
100.0	Salt pork, cooked	22621000
100.0	Fat back, cooked	22621100
100.0	Pork, spareribs, cooked, NS as to fat eaten	22701000
100.0	Pork, spareribs, cooked, lean and fat eaten	22701010
100.0	Pork, spareribs, cooked, lean only eaten	22701020
100.0	Pork, spareribs, barbecued, with sauce, NS as to fat eaten	22701030
100.0	Pork, spareribs, barbecued, with sauce, lean and fat eaten	22701040
100.0	Pork, spareribs, barbecued, with sauce, lean only eaten	22701050
100.0	Pork, cracklings, cooked	22704010

% Pork in Food Item	Food Item Description	USDA food code value
100.0	Pork ears, tail, head, snout, miscellaneous parts, cooked	22705010
100.0	Pork, neck bones, cooked	22706010
100.0	Pork, pig's feet, cooked	22707010
100.0	Pork, pig's feet, pickled	22707020
100.0	Pork, pig's hocks, cooked	22708010
100.0	Pork skin, rinds, deep-fried	22709010
100.0	Pork skin, boiled	22709110
100.0	PORK LIVER, BREADED, FRIED	25110340
100.0	Pork sausage, fresh, bulk, patty or link, cooked	25221410

Table D.8 Food Codes for Egg Items

% Eggs in Food Item	Food Item Description	USDA Food Code
25	Fried egg sandwich	32201000
25	Egg, cheese, and ham on English muffin	32202010
25	Egg, cheese, and ham on biscuit	32202020
25	Egg, cheese and ham on bagel	32202025
25	Egg, cheese, and sausage on English muffin	32202030
25	Egg, cheese, and beef on English Muffin	32202040
25	Egg, cheese, and steak on bagel	32202045
25	Egg, cheese, and sausage on biscuit	32202050
25	Egg, cheese, and sausage griddle cake sandwich	32202055
25	Egg and sausage on biscuit	32202060
25	Egg, cheese, and bacon on biscuit	32202070
25	Egg, cheese, and bacon griddle cake sandwich	32202075
25	Egg, cheese, and bacon on English muffin	32202080
25	Egg, cheese and bacon on bagel	32202085
25	Egg and bacon on biscuit	32202090
25	Egg and ham on biscuit	32202110
25	Egg, cheese and sausage on bagel	32202120
25	Egg and cheese on biscuit	32202200
25	Egg drop soup	32300100
25	Garlic egg soup, Puerto Rican style (Sopa de ajo)	32301100
25	Burrito with eggs, sausage, cheese and vegetables	58100340
25	Burrito with eggs and cheese, no beans	58100350
25	Croissant sandwich with sausage and egg	58127270
25	Croissant sandwich with ham, egg, and cheese	58127310
25	Croissant sandwich with sausage, egg, and cheese	58127330
25	Croissant sandwich with bacon, egg, and cheese	58127350
33	Egg dessert, custard-like, made with water and sugar, Puerto	32120100
66	Egg foo yung (young), NFS	32105200
66	Chicken egg foo yung (young)	32105210
66	Pork egg foo yung (young)	32105220
66	Shrimp egg foo yung (young)	32105230
75	Egg, Benedict	32101500

% Eggs in Food Item	Food Item Description	USDA Food Code
75	Egg, deviled	32102000
75	Egg salad	32103000
100	Egg, whole, raw	31101010
100	Egg, whole, cooked, NS as to cooking method	31102000
100	Egg, whole, boiled	31103000
100	Egg, whole, poached	31104000
100	Egg, whole, fried	31105000
100	Egg, whole, fried without fat	31105010
100	Egg, whole, baked, fat not added in cooking	31106010
100	Egg, whole, baked, fat added in cooking	31106020
100	Egg, whole, pickled	31107000
100	Egg, white only, cooked	31109010
100	Egg, yolk only, raw	31110010
100	Egg, yolk only, cooked	31111010
100	Egg, creamed	32101000
100	Egg omelet or scrambled egg, NS as to fat added in cooking	32104900
100	Egg omelet or scrambled egg, fat not added in cooking	32104950
100	Egg omelet or scrambled egg, fat added in cooking	32105000
100	Egg omelet or scrambled egg, with cheese	32105010
100	Egg omelet or scrambled egg, with fish	32105020
100	Egg omelet or scrambled egg, with ham or bacon	32105030
100	Egg omelet or scrambled egg, with dark-green vegetables	32105040
100	Egg omelet or scrambled egg, with vegetables other than dark	32105050
100	Egg omelet or scrambled egg, with peppers, onion, and ham	32105060
100	Egg omelet or scrambled egg, with mushrooms	32105070
100	Egg omelet or scrambled egg, with cheese and ham or bacon	32105080
100	Egg omelet or scrambled egg, with cheese, ham or bacon, and	32105085
100	Egg omelet or scrambled egg, with potatoes and/or onions (To	32105100
100	Egg omelet or scrambled egg, with beef	32105110
100	Egg omelet or scrambled egg, with sausage and cheese	32105121
100	Egg omelet or scrambled egg, with sausage	32105122
100	Egg omelet or scrambled egg, with hot dogs	32105125

% Eggs in Food Item	Food Item Description	USDA Food Code
100	Egg omelet or scrambled egg, with onions, peppers, tomatoes,	32105130
100	Egg omelet or scrambled egg, with chorizo	32105160
100	Egg omelet or scrambled egg with chicken	32105170
100	Huevos rancheros	32105180
100	Meringues	32401000

Table D.9 Food Codes for Milk Items

% Milk in Food Item	Food Item Description	USDA Food Code
50	Cafe con leche prepared with sugar	11561010
50	Ice cream sandwich	13120500
50	Ice cream cookie sandwich	13120550
50	Ice cream cone with nuts, flavors other than chocolate	13120700
50	Ice cream cone, chocolate covered, with nuts, flavors other	13120710
50	Ice cream cone, chocolate covered or dipped, flavors other t	13120720
50	Ice cream cone, no topping, flavors other than chocolate	13120730
50	Ice cream cone, no topping, NS as to flavor	13120740
50	Ice cream cone with nuts, chocolate ice cream	13120750
50	Ice cream cone, chocolate covered or dipped, chocolate ice c	13120760
50	Ice cream cone, no topping, chocolate ice cream	13120770
50	Ice cream cone, chocolate covered, with nuts, chocolate ice	13120780
50	Ice cream sundae cone	13120790
50	Ice cream soda, flavors other than chocolate	13120800
50	Ice cream sundae, fruit topping, with whipped cream	13121100
50	Ice cream sundae, chocolate or fudge topping, with whipped c	13121300
50	Ice cream pie, no crust	13122100
50	Pudding, bread	13210110
50	Pudding, Mexican bread (Capirotada)	13210180
50	Cheese sandwich	14640000
50	Cheese sandwich, grilled	14640100
50	Cheese, nuggets or pieces, breaded, baked, or fried	14660200
75	Pudding, with fruit and vanilla wafers	13241000
100	Milk, NFS	11100000
100	Milk, cow's, fluid, whole	11111000
100	Milk, calcium fortified, cow's, fluid, whole	11111150
100	Milk, cow's, fluid, other than whole, NS as to 2%, 1%, or sk	11112000
100	Milk, cow's, fluid, 2% fat	11112110
100	Milk, cow's, fluid, acidophilus, 1% fat	11112120
100	Milk, cow's, fluid, acidophilus, 2% fat	11112130
100	Milk, cow's, fluid, 1% fat	11112210
100	Milk, cow's, fluid, skim or nonfat, 0.5% or less butterfat	11113000

% Milk in Food Item	Food Item Description	USDA Food Code
100	Milk, cow's, fluid, lactose reduced, 1% fat	11114300
100	Milk, cow's, fluid, lactose reduced, nonfat	11114320
100	Milk, cow's, fluid, lactose reduced, 2% fat	11114330
100	Milk, cow's, fluid, lactose reduced, whole	11114350
100	Buttermilk, fluid, nonfat	11115000
100	Buttermilk, fluid, 1% fat	11115100
100	Buttermilk, fluid, 2% fat	11115200
100	Milk, goat's, fluid, whole	11116000
100	Yogurt, NS as to type of milk or flavor	11410000
100	Yogurt, plain, NS as to type of milk	11411010
100	Yogurt, plain, whole milk	11411100
100	Yogurt, plain, lowfat milk	11411200
100	Yogurt, plain, nonfat milk	11411300
100	Yogurt, vanilla, lemon, or coffee flavor, NS as to type of m	11420000
100	Yogurt, vanilla, lemon, or coffee flavor, whole milk	11421000
100	Yogurt, vanilla, lemon, maple, or coffee flavor, lowfat milk	11422000
100	Yogurt, vanilla, lemon, maple, or coffee flavor, nonfat milk	11423000
100	Yogurt, vanilla, lemon, maple, or coffee flavor, nonfat milk	11424000
100	Yogurt, chocolate, NS as to type of milk	11425000
100	Yogurt, fruit variety, NS as to type of milk	11430000
100	Yogurt, fruit variety, whole milk	11431000
100	Yogurt, fruit variety, lowfat milk	11432000
100	Yogurt, fruit variety, lowfat milk, sweetened with low-calor	11432500
100	Yogurt, fruit variety, nonfat milk	11433000
100	Yogurt, fruit variety, nonfat milk, sweetened with low-calor	11433500
100	Yogurt, fruit and nuts, lowfat milk	11445000
100	Yogurt, frozen, NS as to flavor, NS as to type of milk	11459990
100	Yogurt, frozen, flavors other than chocolate, NS as to type	11460000
100	Yogurt, frozen, chocolate, NS as to type of milk	11460100
100	Yogurt, frozen, NS as to flavor, lowfat milk	11460150
100	Yogurt, frozen, chocolate, lowfat milk	11460160
100	Yogurt, frozen, flavors other than chocolate, lowfat milk	11460170
100	Yogurt, frozen, NS as to flavor, nonfat milk	11460190

% Milk in Food Item	Food Item Description	USDA Food Code
100	Yogurt, frozen, chocolate, nonfat milk	11460200
100	Yogurt, frozen, flavors other than chocolate, nonfat milk	11460300
100	Yogurt, frozen, chocolate, nonfat milk, with low-calorie swe	11460400
100	Yogurt, frozen, flavors other than chocolate, nonfat milk, w	11460410
100	Yogurt, frozen, flavors other than chocolate, whole milk	11460440
100	Yogurt, frozen, cone, chocolate	11461250
100	Yogurt, frozen, cone, flavors other than chocolate	11461260
100	Yogurt, frozen, cone, flavors other than chocolate, lowfat m	11461270
100	Yogurt, frozen, cone, chocolate, lowfat milk	11461280
100	Milk, chocolate, NFS	11511000
100	Milk, chocolate, whole milk-based	11511100
100	Milk, chocolate, reduced fat milk-based, 2% (formerly "lowfa	11511200
100	Milk, chocolate, skim milk-based	11511300
100	Milk, chocolate, lowfat milk-based	11511400
100	Cocoa, hot chocolate, not from dry mix, made with whole milk	11512000
100	Cocoa and sugar mixture, milk added, NS as to type of milk	11513000
100	Cocoa and sugar mixture, whole milk added	11513100
100	Cocoa and sugar mixture, reduced fat milk added	11513150
100	Cocoa and sugar mixture, lowfat milk added	11513200
100	Cocoa and sugar mixture, skim milk added	11513300
100	Chocolate syrup, milk added, NS as to type of milk	11513400
100	Chocolate syrup, whole milk added	11513500
100	Chocolate syrup, reduced fat milk added	11513550
100	Chocolate syrup, lowfat milk added	11513600
100	Chocolate syrup, skim milk added	11513700
100	Cocoa, whey, and low-calorie sweetener mixture, lowfat milk	11516000
100	Milk beverage, made with whole milk, flavors other than choc	11519000
100	Milk, flavors other than chocolate, whole milk-based	11519050
100	Milk, malted, unfortified, NS as to flavor, made with milk	11520000
100	Milk, malted, unfortified, chocolate, made with milk	11521000
100	Milk, malted, unfortified, natural flavor, made with milk	11522000
100	Milk, malted, fortified, chocolate, made with milk	11526000
100	Milk, malted, fortified, NS as to flavor, made with milk	11527000

% Milk in Food Item	Food Item Description	USDA Food Code
100	Eggnog, made with whole milk	11531000
100	Eggnog, made with 2% reduced fat milk (formerly eggnog, made	11531500
100	Milk shake, homemade or fountain-type, NS as to flavor	11541100
100	Milk shake, homemade or fountain-type, chocolate	11541110
100	Milk shake, homemade or fountain-type, flavors other than ch	11541120
100	Milk shake with malt	11541400
100	Milk shake, made with skim milk, chocolate	11541500
100	Milk shake, made with skim milk, flavors other than chocolat	11541510
100	Milk fruit drink	11551050
100	Orange Julius	11552200
100	Fruit smoothie drink, made with fruit or fruit juice and dai	11553000
100	Fruit smoothie drink, NFS	11553100
100	Chocolate-flavored drink, whey- and milk-based	11560000
100	Flavored milk drink, whey- and milk-based, flavors other tha	11560020
100	Instant breakfast, powder, milk added	11612000
100	Instant breakfast, powder, sweetened with low calorie sweete	11613000
100	Cream, NS as to light, heavy, or half and half	12100100
100	Cream, light, fluid	12110100
100	Cream, light, whipped, unsweetened	12110300
100	Cream, half and half	12120100
100	Cream, half and half, fat free	12120110
100	Cream, heavy, fluid	12130100
100	Cream, heavy, whipped, sweetened	12140000
100	Sour cream	12310100
100	Sour cream, reduced fat	12310300
100	Sour cream, light	12310350
100	Sour cream, fat free	12310370
100	Dip, sour cream base	12350000
100	Dip, sour cream base, reduced calorie	12350020
100	Spinach dip, sour cream base	12350100
100	Ice cream, NFS	13110000
100	Ice cream, regular, flavors other than chocolate	13110100
100	Ice cream, regular, chocolate	13110110

% Milk in Food Item	Food Item Description	USDA Food Code
100	Ice cream, rich, flavors other than chocolate	13110120
100	Ice cream, rich, chocolate	13110130
100	Ice cream, soft serve, flavors other than chocolate	13110200
100	Ice cream, soft serve, chocolate	13110210
100	Ice cream, soft serve, NS as to flavor	13110220
100	ICE CREAM W/ SHERBET	13125100
100	Ice cream, fried	13126000
100	Light ice cream, flavors other than chocolate (formerly ice	13130300
100	Light ice cream, chocolate (formerly ice milk)	13130310
100	Light ice cream, no sugar added, NS as to flavor	13130320
100	Light ice cream, no sugar added, flavors other than chocolat	13130330
100	Light ice cream, no sugar added, chocolate	13130340
100	LIGHT ICE CREAM,PREMIUM, NOT CHOC (FORMERLY ICE MILK)	13130350
100	Light ice cream, soft serve, NS as to flavor (formerly ice m	13130590
100	Light ice cream, soft serve, flavors other than chocolate (f	13130600
100	Light ice cream, soft serve cone, chocolate (formerly ice mi	13130630
100	Light ice cream, soft serve cone, NS as to flavor (formerly	13130640
100	Light ice cream, cone, chocolate (formerly ice milk)	13140550
100	Light ice cream, sundae, soft serve, chocolate or fudge topp	13140660
100	Light ice cream, sundae, soft serve, not fruit or chocolate	13140680
100	LIGHT ICE CREAM,W/ SHERBET OR ICE CREAM (FORMERLY ICE MILK)	13141100
100	Sherbet, all flavors	13150000
100	MILK DESSERT, FROZEN, MADE FROM LOWFAT MILK	13160000
100	MILK DESSERT,FZN,LOWFAT,W/LOW CAL SWEET,NOT CHOC	13160100
100	Fat free ice cream, no sugar added, chocolate	13160150
100	Fat free ice cream, no sugar added, flavors other than choco	13160160
100	MILK DESSERT,FROZEN,LOWFAT,NOT CHOCOLATE	13160200
100	MILK DESSERT, FROZEN, LOWFAT, CHOCOLATE	13160210
100	Fat free ice cream, flavors other than chocolate	13160400
100	Fat free ice cream, chocolate	13160410
100	MILK DSRT,FROZ,MILK-FAT FREE,W/SIMPLESSE, NOT CHOC	13160550
100	MILK DESSERT, FROZ, W/ LOW CAL SWEETENER, NOT CHOC	13160600

% Milk in Food Item	Food Item Description	USDA Food Code
100	MILK DESSERT, FROZ, W/ LOW CAL SWEETENER, CHOCOLATE	13160650
100	Milk dessert sandwich bar, frozen, made from lowfat milk	13161500
100	Milk dessert bar, frozen, made from lowfat milk and low calo	13161600
100	Light ice cream, bar or stick, with low-calorie sweetener, c	13161630
100	Pudding, NFS	13200110
100	Pudding, chocolate, ready-to-eat, NS as to from dry mix or c	13210220
100	Pudding, chocolate, ready-to-eat, low calorie, containing ar	13210250
100	Pudding, flavors other than chocolate, ready-to-eat, NS as t	13210280
100	Pudding, flavors other than chocolate, ready-to-eat, low cal	13210290
100	Custard	13210300
100	Custard, Puerto Rican style (Flan)	13210350
100	Pudding, rice	13210410
100	Pudding, tapioca, made from home recipe, made with milk	13210500
100	Pudding, tapioca, made from dry mix, made with milk	13210520
100	Pudding, coconut	13210610
100	Puerto Rican pumpkin pudding (Flan de calabaza)	13210810
100	Pudding, flavors other than chocolate, prepared from dry mix	13220110
100	Pudding, chocolate, prepared from dry mix, milk added	13220120
100	Pudding, flavors other than chocolate, prepared from dry mix	13220210
100	Pudding, chocolate, prepared from dry mix, low calorie, cont	13220220
100	Mousse, chocolate	13250000
100	Milk dessert or milk candy, Puerto Rican style (Dulce de lec	13252200
100	Barfi or Burfi, Indian dessert, made from milk and/or cream	13252500
100	Tiramisu	13252600
100	Custard pudding, flavor other than chocolate, baby food, NS	13310000
100	Custard pudding, baby food, flavor other than chocolate, str	13311000
100	Custard pudding, baby food, flavor other than chocolate, jun	13312000
100	White sauce, milk sauce	13411000
100	Milk gravy, quick gravy	13412000
100	Cheese, NFS	14010000
100	Cheese, Cheddar or American type, NS as to natural or proces	14010100
100	Cheese, natural, NFS	14100100

% Milk in Food Item	Food Item Description	USDA Food Code
100	Cheese, Blue or Roquefort	14101010
100	Cheese, Brick	14102010
100	Cheese, Brie	14103020
100	Cheese, natural, Cheddar or American type	14104010
100	Cheese, Cheddar or American type, dry, grated	14104020
100	Cheese, Colby	14104200
100	Cheese, Colby Jack	14104250
100	Cheese, Feta	14104400
100	Cheese, Fontina	14104600
100	Cheese, goat	14104700
100	Cheese, Gouda or Edam	14105010
100	Cheese, Gruyere	14105200
100	Cheese, Monterey	14106200
100	Cheese, Monterey, lowfat	14106500
100	Cheese, Mozzarella, NFS	14107010
100	Cheese, Mozzarella, whole milk	14107020
100	Cheese, Mozzarella, part skim	14107030
100	Cheese, Mozzarella, nonfat or fat free	14107060
100	Cheese, Muenster	14107200
100	Cheese, Muenster, lowfat	14107250
100	Cheese, Parmesan, dry grated	14108010
100	Cheese, Parmesan, hard	14108020
100	Cheese, Parmesan, low sodium	14108050
100	Parmesan cheese topping, fat free	14108060
100	Cheese, Provolone	14108400
100	Cheese, Swiss	14109010
100	Cheese, Swiss, low sodium	14109020
100	Cheese, Swiss, lowfat	14109030
100	Cheese, Cheddar or Colby, low sodium	14110010
100	Cheese, Cheddar or Colby, lowfat	14110030
100	Cheese, Mexican blend	14120010
100	Queso Anejo (aged Mexican cheese)	14131000
100	Queso Asadero	14131500

% Milk in Food Item	Food Item Description	USDA Food Code
100	Queso Chihuahua	14132000
100	Queso Fresco	14133000
100	Cheese, cottage, NFS	14200100
100	Cheese, cottage, creamed, large or small curd	14201010
100	Cottage cheese, farmer's	14201200
100	Cheese, Ricotta	14201500
100	Cheese, cottage, with fruit	14202010
100	Cheese, cottage, salted, dry curd	14203020
100	Puerto Rican white cheese (queso del pais, blanco)	14203510
100	Cheese, cottage, lowfat (1-2% fat)	14204010
100	Cheese, cottage, lowfat, with fruit	14204020
100	CHEESE, YOGURT, NFS	14210000
100	Cheese, cream	14301010
100	Cheese, cream, light or lite (formerly called Cream Cheese L	14303010
100	Cheese spread, cream cheese, regular	14420200
100	Cheese, cottage cheese, with gelatin dessert	14610200
100	Topping from cheese pizza	14620300
100	Topping from vegetable pizza	14620310
100	Topping from meat pizza	14620320
100	Cheese fondue	14630100
100	Cheese sauce	14650100
100	Cheese sauce made with lowfat cheese	14650150
100	Alfredo sauce	14650160

Appendix E
Determination of Chemicals for Multipathway Analysis

E.1 Introduction

The AB-2588 program assesses the risk from airborne chemicals that are often emitted by facilities at high temperature and pressure in the presence of particulate matter. Some of these chemicals will be emitted and remain in vapor form. The inhalation cancer risk and noncancer hazard from such volatile chemicals are likely to be much greater than the risk from other possible exposure pathways. Other chemicals, such as semi-volatile organic or metal toxicants, can either be emitted as particles, form particles after emission from the facility, or adhere to existing particles. Some chemicals will partition between the vapor and particulate phases. Some chemicals such as PAHs have been found to have a portion of the particle associated mass in reversible equilibrium with the vapor phase and a portion irreversibly bound (Eiceman and Vandiver, 1983). Chemicals in the particulate phase can be removed from the atmosphere by settling. The settling of smaller particles can be enhanced by coalescence into larger particles with greater mass.

There are a number of exposure pathways by which humans may be exposed to airborne chemicals. Particulate associated chemicals can be deposited directly onto soil, onto the leaves of crops, or onto surface waters. Crops may also be contaminated by root uptake of chemicals. Livestock such as chickens, pigs and cows may be contaminated by inhalation of such chemicals or by consumption of contaminated feed, pasture, or surface waters. Humans may be exposed to these chemicals through inhalation, consumption of crops, soil, surface waters, meat, eggs and dairy products. Infants may be exposed through consumption of human breast milk.

E.2 Criteria for Selection of Chemicals for Multipathway Analysis

Chemicals listed in Appendix A, “Substances for Which Emissions Must be Quantified” which have been previously reported to be emitted by facilities in California under the Air Toxics “Hot Spots” Act were considered as candidates for multipathway analysis. From the chemicals meeting this criteria, chemicals which had been considered in the past to be multipathway chemicals or were thought to be likely candidates were selected for further analysis. We chose chemicals on the basis of whether they might be particle bound.

Junge (1977) developed a theoretical model for the partitioning of the exchangeable fraction of an airborne chemical between the vapor and particulate phases in the ambient air.

$$\theta = \frac{bS^{(p)}}{P^{s}_{L} + bS^{(p)}}$$

Where:

θ = fraction of the total mass of chemical on the particle phase (unitless)
b = a constant (mm Hg cm^3/cm^2)
$S^{(p)}$ = total surface area of particle per unit volume of air (cm^2/cm^3)
P^{s}_{L} = saturation pressure of the liquid chemical at ambient temperature (mm Hg)

Junge (1977) did not distinguish between solid and liquid phase vapor pressures. Pankow (1987) recognized the importance of using the liquid phase vapor pressure. When the chemical of interest is a solid at the temperature of interest, the subcooled liquid vapor pressure must be used. The subcooled liquid vapor pressure is an extrapolation of the saturated liquid vapor pressure below the melting point where the compound actually exists as solid (Boethling and McKay, 2000). The subcooled liquid vapor pressure can be estimated using the following equation:

$$P^{s}_{L}/P^{s}_{s} = \exp[\Delta S_f (T_m - T)/RT]/RT$$

Where:

P^{s}_{L} = sub cooled liquid vapor pressure of the liquid chemical at ambient temperature (Pascal).
P^{s}_{s} = saturated vapor of the solid at room temperature
ΔS_f = entropy of fusion (J/mol K)
T_m = melting point temperature (K)
T = ambient temperature (K)
R= gas constant (8.3143 joules/K mole)

Values for ΔS_f may be obtained in the literature. In cases where a literature value is not available a default value of 56.45 has been suggested by Boethling and McKay (2000).

The percentage of the total mass of chemical (vapor plus particulate fractions) is determined by multiplying θ times 100. The percentage of the total mass of chemical that is in particulate phase is determined in part by the concentration of particles in the air. For our purposes, we used an average concentration of particles in urban air determined by Whitby (1978). The concentration of particles was 1.04×10^{-4} $\mu g/cm^3$. The surface area per μg of particle was assumed to be 0.05 $cm^2/\mu g$. Thus the $S^{(p)}$ is calculated to be 5.2×10^{-6}

cm^2/cm^3. The value of b used is the default value of 0.1292 mm Hg cm^3/cm^2 recommended by Pankow (1987).

It should be noted that the particle bound associated fraction of some semi-volatile organic toxicants has been found to consist of a non-exchangeable fraction and a fraction which equilibrates with the vapor phase (Bidleman and Foreman, 1987). The equation of Junge (1977) only addresses the exchangeable fraction. This means that the actual fraction of the total mass that is particle bound material may be somewhat higher than the theoretical model which Junge (1977) proposed. The partitioning of semi-volatile organic toxicants between the vapor phase and particles has been experimentally investigated by Bidleman et al. (1986) and Bidleman and Foreman (1987). High volume sampling has been done in several cities in which the particulate and vapor fractions have been collected on filters and adsorbents. This work has supported the validity of the theoretical model of Junge (1977).

The Junge (1977) and Pankow (1987) model appears to be the best available to develop criteria to determine which chemicals emitted by facilities in the AB-2588 program should undergo multipathway analysis. The liquid or subcooled liquid vapor pressure at ambient temperatures determines the fraction of chemical that will be particle associated. The vapor pressure is available for most of the chemicals for which the determination needs to be made.

It should be noted that the Junge (1977) model was designed to look at the partitioning of chemicals between the particle and vapor phases under equilibrium conditions in the atmosphere. The initial conditions under which particle formation may occur as chemicals are emitted into the atmosphere may be different from the conditions assumed by Junge (1977). The chemicals of concern in the AB-2588 program may be emitted at high temperatures and pressures in the presence of a high concentration of particulate matter. Such conditions may favor partitioning of mass toward the particulate fraction. It is also possible that such conditions might favor the formation of a greater fraction of non-exchangeable particle associated chemical which is not taken into account in the Junge (1977) equation. The rapid cooling from high temperature to ambient temperature may also influence the percent of total mass which is particle bound in ways that are not accounted for in the simple equilibrium model of Junge (1977).

OEHHA has decided that chemicals with less than 0.5% of the total mass in the particle-associated fraction will not be considered for multipathway analysis. The 0.5% is a relatively small percentage of the total mass. This percentage was chosen in part to compensate for the uncertainties involved in extrapolation of the Junge (1977) model to the conditions under which particles may be formed in the stacks of facilities. Thus chemicals with vapor pressures greater than 1.34×10^{-4} mm Hg at 25° C will not be considered for multipathway analysis. An exception to this rule is the inclusion of hexachlorobenzene (HCB) for multipathway

analysis, even though its calculated percentage of total mass in the particulate phase is expected to be below 0.5%. The criteria for including HCB is discussed in Section E.3 below. It should be noted that the chemicals for which noninhalation pathway risks are a significant fraction of the total risk are metals, PAH's, PCB's, polychlorinated dibenzo-p-dioxins and furans. These chemicals have much higher percentages of total mass in the particulate fraction than 0.5%.

There are some toxic compounds without measurable vapor pressure at 25°C such as the metals and their compounds. These metals include lead, mercury compounds, nickel, selenium, fluoride, beryllium, arsenic, chromium VI and cadmium. These toxicants are included on the list of chemicals for multipathway analysis.

In Table E.1 we have calculated the air/particle partition coefficients of the compounds emitted by facilities for which it appeared possible that a significant fraction of the total mass could be in the particulate fraction. In some cases the saturated vapor pressure at a temperature at or near ambient temperature (25°C) is not available, the air/particle coefficient canould not be calculated using modern tools such as USEPA's SPARC.

For PAHs, consideration for multipathway analysis is largely confined to PAHs with 4 or more fused rings because a significant fraction of their total mass is in the particle phase. Naphthalene contains 2 fused rings and is included in the Hot Spots program as a carcinogen. However, it does not have a significant percentage of its total mass in the particle phase, so is not considered for multipathway analysis. The PAHs with 3 fused rings (e.g., phenanthrene, fluorine, acenaphthene) are also predominantly found in gaseous form and the data are currently too limited or inadequate to list any of them as carcinogens. Laboratory studies of sludge-amended soils containing PAHs have also shown significant loss through volatilization only for PAHs with less than 4 fused rings (Wild and Jones, 1993). Thus, speciated analysis for PAHs that include only the compounds with 4 or more fused rings can be used for multipathway assessment.

E-3 The Octanol-Water Coefficient as a Means of Determining Gas-Particle Partitioning.

A number of researchers have investigated the use of the octanol-water coefficient as a predictor of gas particle partitioning in the environment. At least some of the research has involved looking at gas-particle partitioning in the urban environment under equilibrium conditions where there were existing particles from a variety of sources (e.g. diesel exhaust, road dust). Existing particles are thought to have a lipid bilayer into which gaseous chemicals can equilibrate. There is some question whether chemicals emitted from a stack would have time to interact with existing urban particles before reaching nearby receptors.

Particulate matter in the air around facilities may not be present in very high concentrations. The Junge (1977) model is a simple equilibrium model.

In the past 15 years, there have been advances in the understanding of the organics and semi-volatile organic compounds partitioning between the gas phase and the organic condensed phase on the airborne particles and the gas phase, using the octanol/air partition coefficient (K_{OA}). Because the equation for estimating partitioning involves the octanol/air partition coefficient (K_{OA}), Tthis model is referred to as the K_{OA} absorption model, while the Junge-Pankow is anknown as an adsorption model. Several studies have described the octanol/air partition coefficients for chlorobenzenes, PCBs, DDT, PAHs and polychlorinated naphthalenes (PCNs) (Harner and MacKay, 1995; Komp and McLachlan,1997; Harner and Bidleman, 1998).

K_{OA} is defined as $K_{OA} = C_o/C_A$, where C_o (mol/L) is the concentration of the compound in 1-octanol and C_A (mol/L) is the gaseous concentration at equilibrium. For the calculation, K_{OA} can be derived as $K_{OA} = K_{OW}/K_{AW} = K_{OW}RT/H$, where K_{OW} is the octanol/water partition coefficient, K_{AW} is the air/water partition coefficient, H is the Henry's Law constant (J/mol), R is the ideal gas constant (J/mol/K), and T is the absolute temperature (K) (Komp and McLachlan, 1997).

The particle/gas partition coefficient (K_P) is defined as $K_P = C_p/C_g$, where C_p is the concentration on particles (ng/µg of particles), and C_g is the gas-phase concentration (ng/m^3 of air) (Harner and Bidleman, 1998). The relation between K_P and K_{OA} is defined as:

$$\log K_P = \log K_{OA} + \log f_{om} - 11.91$$

where, f_{om}- is the= organic matter fraction of the particles.

The fraction (ø) of compound in the particle phase is

$$ø = K_P (TSP) / [1 + K_P (TSP)]$$

where, TSP is the= total suspended particle concentration.

Using f_{om} = 20% (Harner and Bidleman, 1998) and the afore-mentioned average concentration of particles in urban air determined by Whitby (1978), TSP = 1.04 X 10^{-4} µg/cm^3 = 104 µg/m^3 , we obtained the percentage of compound on particles (ø x 100) for selected chemicals through the K_{OA} absorption model, presented as the last column in Table E.1 below. The values compare well with those obtained through Junge-Pankow adsorption model.

Table E1 Calculation of Air/Particle Coefficients and Percent of Particle Associated Total Mass for Selected Chemicals.

Chemical	Vapor Pressure (mm Hg)	Temp. (°C)	Ref. (Vapor Press.)	Air/Particle Partition Coefficient (θ)	% Particulate (of total mass)	% Particulate (K_{OA} model)
4,4-Methylene dianiline	1.0	197	1	NA	NA	31.5
o-Cresol	0.28*	38.2,	2	$2.44X10^{-6}$	2.44×10^{-4}	4.65×10^{-3}
m-Cresol	0.39**	25	2	$1.71x10^{-6}$	$1.71x10^{-4}$	6.64×10^{-3}
p-Cresol	0.37**	25	2	$1.81x10^{-6}$	$1.81x10^{-4}$	5.45×10^{-3}
Cellosolve	5.63***	25	3	$1.19x10^{-7}$	$1.19x10^{-5}$	6.38×10^{-5}
Cellosolve acetate	2.12***	25	3	$3.17x10^{-7}$	$3.19x10^{-5}$	3.40×10^{-5}
Mercury (elemental)	$1.20x10^{-3}$***	25	4	$5.6x10^{-4}$	0.056	NA****
Hexachlorocyclo-hexanes (Lindane)	$1.18 x10^{-4}$**	20	5	$5.66x10^{-3}$	0.57	6.39×10^{-2}
Phthalates						
Diethylhexylphthalate	$1.97 X10^{-7}$***	25	3	$7.73x10^{-1}$	77.3	98.9
Chlorobenzenes						
Chlorobenzene	12.2***	25	6	$5.53x10^{-8}$	$5.53x 10^{-6}$	1.09×10^{-5}
p-Dichlorobenzene	0.65***	25	6	$1.03x10^{-6}$	$9.93x10^{-5}$	9.96×10^{-5}
m-Dichlorobenzene	2.30***	25	6	$1.03x10^{-6}$	$1.03x10^{-4}$	4.24×10^{-5}
o-Dichlorobenzene	0.39***	25	6	$1.71x10^{-6}$	$1.71x10^{-4}$	6.53×10^{-5}
1,2,3-Trichlorobenzene	0.39*	40	6	$1.71x10^{-6}$	$1.71x10^{-4}$	3.30×10^{-4}
1,2,4-Trichlorobenzene	0.45*	38	6	$1.48x10^{-6}$	$1.48 x10^{-6}$	2.88×10^{-4}
1,2,3,4-Tetrachlorobenzene	6.58×10^{-2}*		6	$1.02x10^{-5}$	$1.02x10^{-3}$	1.39×10^{-3}
1,2,3,5-Tetrachlorobenzene	0.14*		6	$4.82x10^{-6}$	$4.82x10^{-4}$	3.41×10^{-4}
Pentachlorobenzene	6.67×10^{-3}*	25	6	$1.01x10^{-4}$	$1.01x10^{-2}$	7.36×10^{-3}

Chemical	Vapor Pressure (mm Hg)	Temp. (°C)	Ref. (Vapor Press.)	Air/Particle Partition Coefficient (θ)	% Particulate (of total mass)	% Particulate (K_{OA} model)
Hexachlorobenzene	2.96 x 10^{-4}*	25	6	2.96x10^{-4}	2.96x10^{-2}	1.53 x 10^{-2}
PAHs						
Naphthalene (2 fused rings)	0.31*	25	7	2.14x10^{-6}	2.14x10^{-4}	3.46 x 10^{-4}
Acenaphthene (3 fused rings)	3.02 x10^{-3}*	25	7	2.23x10^{-5}	2.23x10^{-3}	4.34 x 10^{-3}
Acenaphthylene (3 fused rings)	6.67 x10^{-3}	25	7	1.00x10^{-4}	0.01	7.55 x 10^{-3}
Anthracene (3 fused rings)	4.2 x10^{-6}*	25	7	1.57x10^{-2}	1.57	6.78 x 10^{-2}
Benzo[a]anthracene (4 fused rings)	4.07 x10^{-6}*	25	7	1.42x10^{-1}	14.2	8.15
Chrysene (4 fused rings)	8.81x10^{-8}**	25	7	8.84x10^{-1}	88.4	4.82 x 10^{-5}
Benzo[a]pyrene (5 fused rings)	9.23 x10^{-8}	25	7	8.79x10^{-1}	87.9	60.2
Benzo[b]fluoranthene (5 fused rings)	1.59 x10^{-7}	25	7	8.09x10^{-1}	80.9	NA****
Benzo[k]fluoranthene (5 fused rings)	3.7 x10^{-8}*	25	7	9.48x10^{-1}	94.8	79.9
Dibenz[a,h]-anthracene (5 fused rings)	6.07 x10^{-11}**	25	7	1.00x10^{0}	100	NA****
Indeno[1,2,3cd]-pyrene (6 fused rings)	1.19 x10^{-9}**	25	8	9.98x10^{-1}	99.8	NA****
Chlorophenols						
Pentachlorophenol	1.73x10^{-3}*	25	2	3.88x10^{-4}	3.88x10^{-2}	76.9
2,4,6-Trichlorophenol	2.8x10^{-02}*	25	2	2.34x10^{-5}	2.34x10^{-3}	NA****
2,4,5-Trichlorophenol	4.59x10^{-02}*	25	2	1.46x10^{-5}	1.46x10^{-3}	NA****
Nitrosoamines						
N-Nitrosodiethylamine	8.60x10^{-1}***	20	1	7.81x10^{-7}	7.81x10^{-5}	2.67 x 10^{-5}
N-Nitroso-dimethylamine	8.1***	20	2	8.29x10^{-8}	8.29x10^{-6}	NA****
N-Nitroso-diphenylamine	4.12x10^{2}**	25	2	1.63x10^{-9}	1.63 x10^{-7}	NA****

Chemical	Vapor Pressure (mm Hg)	Temp. (°C)	Ref. (Vapor Press.)	Air/Particle Partition Coefficient (θ)	% Particulate (of total mass)	% Particulate (K_{OA} model)
N-Nitrosodi-n-butylamine	$3.0x10^{-2}$***	20	9	$2.24x10^{-5}$	$2.24x10^{-3}$	NA****
N-Nitrosodi-n-propylamine	$4.15x10^{-1}$***	20	2	$1.62x10^{-6}$	$1.62x10^{-4}$	2.75×10^{-4}
N-Nintrosopyrrolidine	$7.2x10^{-02}$***	20	9	$9.2x10^{-6}$	$9.2x10^{-4}$	NA****
PCBs						
Aroclor 1016	$1.50x10^{-3}$*	25	6	$4.48x10^{-4}$	$4.48x10^{-2}$	1.63×10^{-3}
Aroclor 1221	$1.50x10^{-2}$*	25	6	$4.48x10^{-5}$	$4.48x10^{-03}$	6.53×10^{-4}
Aroclor 1232	$4.05x10^{-3}$***	25	6	$1.66x10^{-4}$	0.17	2.84×10^{-3}
Aroclor 1242	$4.13x10^{-4}$***	25	6	$1.63x10^{-4}$	0.16	1.13×10^{-2}
Aroclor 1248	$3.33x10^{-4}$***	25	6	$1.66x10^{-3}$	0.17	5.17×10^{-2}
Aroclor 1254	$7.73x10^{-5}$***	25	6	$8.62x10^{-3}$	0.86	0.142
Aroclor 1260	$4.40x10^{-6}$***	25	6	$1.32 x10^{-1}$	13.2	1.23
Dioxins and Furans						
2,3,7,8 Tetrachloro-dibenzo-p-dioxin	$4.5x10^{-7}$*	20	7	$5.97x10^{-1}$	59.7	10.7
2,3,7,8 Tetrachloro-dibenzofuran	$9.21x10^{-7}$*	25	7	$9.97x10^{-1}$	99.7	5.18
1,2,3,4,7 Pentachloro-dibenzodioxin	$5.9x10^{-7}$**	25	7	$5.42x10^{-1}$	54.2	85.7
2,3,4,7,8 Pentachloro-dibenzofuran	$1.63x10^{-7}$*	25	7	$4.22x10^{-1}$	42.2	28.4
1,2,3,4,7,8 Hexachlorodibenzo-p-dioxin	$5.89x10^{-9}$*	25	7	$9.17x10^{-1}$	91.7	78.7
1,2,3,4,7,8 Hexachloro-dibenzofuran	$6.07x10^{-8}$*	25	7	$9.89x10^{-1}$	98.9	30.4
1,2,3,4,6,7,8 Heptachlorodibenzo-p-dioxin	$7.68x10^{-9}$*	25	7	$9.76x10^{-1}$	97.6	83.3
1,2,3,4,6,7,8 Heptachloro-dibenzofuran	$1.68x10^{-8}$*	25	7	$9.76x10^{-1}$	97.6	52.8
1,2,3,4,7,8,9 Heptachloro-	$9.79x10^{-9}$*	25	7	$9.87x10^{-1}$	98.7	NA****

Chemical	Vapor Pressure (mm Hg)	Temp. (°C)	Ref. (Vapor Press.)	Air/Particle Partition Coefficient (θ)	% Particulate (of total mass)	% Particulate (K_{OA} model)
dibenzofuran						
1,2,3,4,5,6,7,8 Octachloro-dibenzofuran	$1.95x10^{-9}$*	25	7	$9.97x10^{-1}$	99.7	97.1
1,2,3,4,5,6,7,8 Octachlorodibenzo-p-dioxin	$2.08x10^{-9}$*	25	7	$9.97x10^{-1}$	99.7	93.6

1. IARC, 1986; 2. McKay et al. 1992a; 3. McKone et al., 1993; 4. Cohen et al., 1994; 5. ATSDR, 2005; 6. McKay et al., 1992b; 7. McKay et al., 1992c; 8. Montgomery and Welkom, 1990; 9. Klein, 1982

*Indicates subcooled liquid vapor pressure

**Indicates subcooled liquid vapor pressure estimated according to Boethling and McKay, 2000, page 238.

***Indicates Psat liquid (substance is a liquid at 25 °C)

****Because Kow and/or Henry's Law constant not available

For the nitrosamines, we were not able to locate saturated vapor pressures for N-nitrosomethylethylamine, N-nitrosomorpholine, and N-nitrosopiperidine. We were able to find saturated vapor pressures for N-nitrosodiethylamine, N-nitrosdimethylamine, N-nitrosodiphenylamine, N-nitrosodi-n-butylamine, N-nitrosodi-n-propylamine and N-nitrosopyrrolidine. None of these compounds had particle associated percentages above 0.5%. N-nitrosopyrrolidine was structurally similar to N-nitrosomorpholine and N-nitrosopiperidine. N-nitrosopyrrolidine has a particle associated percentage of 9.2×10^{-4}. This is well below the 0.5% that we selected as our cutoff. We therefore felt that N-nitrosomorpholine and N-nitrosopiperidine were unlikely to have a particle bound percentage above 0.5% and thus we excluded these compounds from multipathway consideration. N-nitrosomethylethylamine did not appear likely to have a particle bound percentage above N-nitrosodiethylamine, N-nitrosodimethylamine or N-nitrosodi-n-butylamine. All of these nitrosamines are well below the 0.5% cutoff.

Table E2. Chemicals for Which Multipathway Risks Need to be assessed.

4,4'-methylene dianiline[1]
creosotes
diethylhexylphthalate
hexachlorobenzene
hexachlorocyclohexanes
PAHs (including but not limited to the following:)[2]

- benz[a]anthracene
- benzo[b]fluoranthene
- benzo[j]fluoranthene
- benzo[k]fluoranthene
- benzo[a]pyrene
- dibenz[a,h]acridine
- dibenz[a,j]acridine
- 7H-dibenzo[c,g]carbazole
- 7,12-dimethylbenz[a]anthracene
- 3-methylcholanthrene
- 5-methylchrysene
- dibenz[a,h]anthracene
- dibenzo[a,e]pyrene
- dibenzo[a,h]pyrene
- dibenzo[a,i]pyrene
- dibenzo[a,l]pyrene
- chrysene
- indeno[1,2,3-cd]pyrene

PCBs[3]

Polychlorinated dibenzo-p-dioxins {PCDDs} (including but not limited to the following, but excluding dioxins with less than four chlorines:)[4]

- 2,3,7,8 tetrachlorodibenzo-p-dioxin
- 1,2,3,7,8 pentachloro-p-dioxin
- 1,2,3,4,7,8 hexachlorodibenzo-p-dioxin
- 1,2,3,6,7,8 hexachlorodibenzo-p-dioxin
- 1,2,3,7,8,9 hexachlorodibenzo-p-dioxin
- 1,2,3,4,6,7,8 heptachlorodibenzo-p-dioxin
- 1,2,3,4,5,6,7,8 Octachlorodibenzo-p-dioxin

Polychlorinated dibenzofurans {PCDFs} (including but not limited to the following, but excluding dibenzofurans with less than four chlorines:)[4]

Table E2. Chemicals for Which Multipathway Risks Need to be Assessed (Cont.).

2,3,7,8 tetrachlorodibenzofuran
1,2,3,7,8 pentachlorodibenzofuran
2,3,4,7,8 pentachlorodibenzofuran
1,2,3,4,7,8 hexachlorodibenzofuran
1,2,3,6,7,8 hexachlorodibenzofuran
1,2,3,7,8,9 hexachlorodibenzofuran
2,3,4,6,7,8 hexachlorodibenzofuran
1,2,3,4,6,7,8 heptachlorodibenzofuran
1,2,3,4,7,8,9 heptachlorodibenzofuran
1,2,3,4,5,6,7,8 Octachlorodibenzofuran

arsenic and arsenic compounds
beryllium and beryllium compounds
cadmium and cadmium compounds
soluble compounds of chromium VI
fluoride and soluble fluoride compounds
lead and inorganic lead compounds
inorganic mercury compounds
nickel and nickel compounds
selenium and selenium compounds

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[1] The saturated vapor pressure at 25°C or close to 25°C is not available to our knowledge. The other evidence available, a melting point of 91.5°C and a boiling point of 398-399 °C (Merck, 1989) indicate that it is very likely that a very significant fraction of the chemical emitted into the air would be in the particulate phase. In addition the vapor pressure at 197 °C is only 1 mm (IARC, 1986).

[2] PAHs with four or more fused rings (Table E2) are to be assessed for multipathway analysis. If PAH mixtures are reported, instead of specific PAHs, then the cancer potency of the entire mixture should be treated the same as benzo(a)pyrene / 10. If the proportion of a PAH mixture greater than 3 fused rings are known in the mixture but not speciated, then the cancer potency should be treated the same as benzo(a)pyrene.

[3] PCBs is inclusive of all Aroclor mixtures. The information in Table E1 indicates that some of the Aroclor mixtures do not have significant air/particle coefficients. However, it is difficult to determine vapor pressures on mixtures of compounds. OEHHA therefore is proposing to include all of the Aroclors in the list of chemicals for multipathway analysis. The percentage of some individual PCBs in the particulate phase has been measured in air samples (Horstmann and McLachlan, 1998). The particulate phase of tetrachlorinated PCBs (PCB 152)

can be expected to be around 1.4%, and increasing to 11.3% for the heptachlorinated PCBs (PCB 180)

[4] From OEHHA analysis (Table E1), it is clear that all polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans should be included in the multipathway analysis.

Table E3 Specific Pathways to be Analyzed for Multipathway Chemicals

Chemical	Soil Ingestion.	Dermal	Meat, Milk & Egg Ingest	Fish Ingestion	Exposed Veg. Ingest.	Leafy Veg. Ingest.	Protected. Veg. Ingest.	Root Veg. Ingest.	Water Ingest	Breast Milk Ingestion.
4,4'-methylene dianiline	X	X		X	X	X			X	
Creosotes	X	X	X	X	X	X			X	
Diethylhexylphthalate	X	X	X	X	X	X			X	
Hexachlorocyclohexanes	X	X	X	X	X	X			X	
Hexachlorobenzene	X	X	X	X	X	X			X	
PAHs	X	X	X	X	X	X			X	X
PCBs	X	X	X	X	X	X			X	X
Dioxins & furans	X	X	X	X	X	X			X	X
Cadmium & compounds	X	X	X	X	X	X	X	X	X	
Chromium VI & compounds	X	X	X	X	X	X	X	X	X	
Inorganic arsenic & cmpds	X	X	X	X	X	X	X	X	X	
Beryllium & compounds	X	X	X	X	X	X	X	X	X	
Lead & compounds	X	X	X	X	X	X	X	X	X	X
Inorganic mercury cmpds	X	X	X	X	X	X	X	X	X	
Nickel & compounds	X	X	X	X	X	X	X	X	X	
Fluoride & compounds	X	X	X	X	X	X	X	X	X	
Selenium and compounds	X	X	X	X	X	X	X	X	X	

OEHHA is recommending that all of the chemicals chosen for multipathway analysis be included in the soil ingestion and dermal pathways. The soil t1/2 values needed to determine concentration in the soil are found in Appendix G. The variates need for the dermal pathway are found in Chapter 6 and Appendix F.

The meat (beef, chicken, pork), cow's milk and egg pathways are listed in one column because the lipid solubility and half life in the body are common factors which determine if these compounds will be present in these three pathways in appreciable concentrations in the fat of meat, milk and eggs.

E.3 Evidence for Inclusion of Hexachlorobenzene for Multipathway Assessment

In the previous Hot Spots Guidance document, semi-volatile substances with less than 0.5% of their total mass in the particle-associated fraction was not considered for multipathway analysis. Although this is a reasonable cut-off for semi-volatile substances predominantly in the gas phase, an exception is made for hexachlorobenzene (HCB). From Table E1, the Junge model shows HCB with a particle/gas ratio of only 0.0296% at 25 ^{o}C. Normally, this would exclude HCB from multipathway analysis. However, actual field measurements of the air/particle partitioning of HCB in Table E.4 shows that the compound is often found in particle form above 0.5%.

The greater than expected particle fraction for HCB is a likely result of environmental conditions at the locations assessed for HCB. The adsorption of HCB on aerosols and subsequent deposition depends on the vapor pressure, the amount and surface area of aerosol particles, and the relevant environmental temperature (Ballschmiter and Wittlinger, 1991). Colder temperatures and greater airborne particulate levels would increase the particle/gas ratio of HCB. In fact, Ballschmiter and Wittlinger (1991) suggested that the particle fraction found at -8 ^{o}C (3.5%) in a rural region will be similar to the particle fraction in urban areas with higher particulate levels and an air temperature of 15 ^{o}C.

Table E.4. Field study vapor/particle distributions of HCB

Study	Particle fraction Concentration (% particle)	Gas phase Concentration (% gas)
Popp et al., 2000[a] Leipzig area Roitzsch area Greppin area	 0.8 pg/Nm^3 (0.9%) 0.5 pg/Nm^3 (0.3%) 2.6 pg/Nm^3 (0.9%)	 83.1 pg/Nm^3 (99.1%) 145.6 pg/Nm^3 (99.7%) 280.6 pg/Nm^3 (99.1%)
Horstmann and McLachlan, (1998)[b]	0.43 pg/m^3 (0.2%)	210 pg/m^3 (99.8%)
Lane et al., 1992[c] Turkey lake Pt. Petre	 3 pg/m^3 (4.1%) 2 pg/m^3 (2.8%)	 71 pg/m^3 (95.9%) 69 pg/m^3 (97.2%)
Ballschmiter and Wittlinger, 1991[d]	4 pg/m^3 (3.5%)	110 pg/m^3 (96.5%)
Bidleman et al., 1987[e] 20 ^{o}C 0 ^{o}C	 (nd)[f] (0.1%) (nd) (0.7%)	 (nd) (99.9%) (nd) (99.3%)

[a] Air samples collected near chlorobenzene-contaminated sites of Bitterfeld region in Germany over a two-week period during the summer of 1998.
[b] Air samples collected over one year in a forest clearing in Germany from May 1995 to April 1996.
[c] Air samples collected during spring, summer, and fall of 1987 in rural regions of Ontario, Canada.
[d] Air sample taken at a mean ambient temperature of -8 ^{o}C outside a small village near a major road in Germany
[e] Data collected from Stockholm, Denver and Columbia. Vapor phase component possibly overestimated due to volatilization (blowoff) from the particle phase in the sampler.
[f] No concentration data was provided.

In addition, Foreman and Bidleman (1987) have suggested that field measurements of HCB particle fractions may be greater than in laboratory settings because sources in the environment includes combustion-derived HCB particle incorporation. Similar to dioxins, combustion of organic material that includes chlorinated substances has been suggested as a primary source of HCB.

Nevertheless, the minor particle fraction of the HCB results in Table E.4 may still not be sufficient to support a multipathway analysis. However, when the extreme environmental persistence of this compound relative to other predominantly gaseous semi-volatile substances (i.e., nitrosamines and chlorophenols) is taken into account, it appears that even a fraction of the compound depositing in the particle bound phase could result in measurable levels in sediment and soil with possible accumulation over time. Field studies at Lake Superior, a relatively pristine water body in which organics deposit primarily from atmospheric sources, have found that HCB accumulated in water, sediment and fish tissue samples

(Eisenreich et al., 1981). In particular, the strong retention of HCB to sediment particulates in the water allowed much of the historical burden to become immobilized in bottom sediments, with a concomitant reduction in the levels of HCB found in the surface waters.

More evidence for HCB's persistence in soil was observed in a laboratory study. Arial application of HCB in a greenhouse with simulated pasture conditions showed that HCB volatilized fairly rapidly from plant and soil surfaces (Beall, 1976). Only 3.4% of HCB remained in the top 2 cm of soil 19 months after spraying. Residues on the grass grown in the soil volatilized considerably faster, with only 1.5% remaining on the plants after two weeks, and <0.01% at 19 months. However, no significant reduction in HCB was found in the deeper 2-4 cm layer of soil after 19 months, showing HCB to be persistent within the soil, including a resistance to microbial degradation and leaching. The immobilization of HCB within the soil is due to its high Kow, leading to strong adsorption to the soil organic fraction.

E.4 Summary

The theoretical model of Junge (1977) uses the liquid or subcooled liquid vapor pressure to determine the percentage of the total airborne mass of chemical that is particulate. Chemicals with 0.5% of the total mass or more in the particulate fraction at 25°C are considered by OEHHA to be multipathway chemicals. This corresponds to toxicants with a vapor pressure of 1.34×10^{-6} mm Hg. A list of multipathway chemicals for the AB-2588 program is provided in Table E2. The percentage of the total mass in the particulate phase and the air/particle partition coefficients for these chemicals and a few other selected chemicals are presented in Table E1.

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Appendix F
Dermal Exposure to Soil-Bound Hot Spots Multipathway Chemicals: Fractional Absorption (ABS) Values

F.1 Introduction

The absorbed dose resulting from dermal exposure to soil-bound chemicals depends on many factors. An algorithm that describes the uptake of chemicals from soil as a function of exposure duration, exposure frequency, chemical concentration in the soil, soil loading, surface area, body weight, averaging time, and fractional absorption (ABS) is discussed in Chapter 6. The purpose of this appendix is to summarize the derivation of the ABS for the "Hot Spots" multipathway chemicals and present the information used in the development of each chemical ABS. A general discussion of the diverse factors influencing dermal absorption of soil-bound chemicals is presented below preceding the chemical ABS summaries.

A small subset of organic and metallic compounds evaluated under the Hot Spots program is subject to deposition onto soil, plants and water bodies. Therefore, exposure can occur by pathways other than inhalation. These chemicals are semi-volatile or nonvolatile, and are therefore partially or wholly in the solid or liquid phase after being emitted. Fate and transport of the deposited chemical must then be estimated in order to assess the impact on soil, water and foods that humans come in contact with. The basis for the selection of these compounds as "Hot Spots" multipathway substances can be found in Appendix E. The organic compounds of relevance listed under the "Hot Spots" program include 4,4'-methylene dianiline, hexachlorocyclohexanes, di(2-ethylhexyl)phthalate (DEHP), polychlorinated dibenzodioxins and dibenzofurans (PCDD/Fs), polychlorinated biphenyls (PCBs) and polycyclic aromatic hydrocarbons (PAHs). The metal or metalloid compounds of relevance include the inorganic salts of arsenic, beryllium, cadmium, fluoride, mercury, lead, nickel, selenium and hexavalent chromium.

F.1.1 Point Estimate Approach for ABS Derivation

An ABS is a chemical-dependent, scenario-dependent value that can vary with the characteristics of the soil matrix and the exposed population. Such characteristics include the relative lipophilicity/hydrophilicity of the compound, soil organic content, soil particle size, soil aging of the chemical, residence time on the skin, and exposed surface area. Some of these issues are discussed in greater detail in Chapter 6. The data necessary to characterize the variability in these variates are often not available. For this reason, the ABS values derived in this document are point estimates. In particular, site specific information on soil organic content and soil particle range are not available. These factors can have a significant impact on chemical absorption from soil and the uncertainty in the dose estimate from dermal absorption because of these and other factors can be large.

To derive a point estimate for a chemical, typically the value from the best and sometimes only study available was selected. If multiple studies were available

with data collected under similar conditions, the most comprehensive study was selected. Or if the studies were of equal reliability, their absorption values would be averaged for ABS determination. In some cases experimental data are not even sufficient for a point estimate ABS and a default ABS is recommended (see below).

F.1.2 Skin Morphology and Dermal Absorption Issues for ABS Determination

The transepidermal uptake of chemicals across skin involves a complex process of transport from the soil matrix to the external protective skin layer called the epidermis, and then through the epidermis to the underlying dermis. The outermost layer of the epidermis is called the stratum corneum, which is thought to provide the major barrier to the absorption of most substances deposited onto the skin surface. The stratum corneum in humans varies in thickness from about 5 µm to over 400 µm on the palms and soles of the feet (Poet and McDougal, 2002; Hostynek, 2003). Below lies the viable epidermis, about 50-100 µm thick, containing keratinocytes that proliferate and differentiate while moving upwards and replacing the stratum corneum cells as they wear away. Below the epidermis lies the hydrous tissue of the dermis perfused by the blood and lymphatic circulation.

Skin appendages, including hair follicles and sweat ducts, transit through all these layers and may provide an alternate pathway for dermal diffusion of some ions such as metal salts (Tregear, 1966; Flynn, 1990). However, skin appendages occupy only a fraction of the surface area of the skin, which may limit their potential as a major diffusion pathway into the systemic circulation.

During the transport through the viable-epidermal and dermal layers, metabolism may also play a role in the absorption process (Kao and Carver, 1990). Metabolism in the dermal layers could also activate a toxicant, resulting in skin as a target organ or producing toxicity elsewhere following systemic absorption. As noted above, specific dermal ABSs for soil-bound chemicals are difficult to obtain due in part to the complex multiphasic nature of the system and lack of published absorption data. Hawley (1985) suggested a default factor of 15 percent to correct for the effect of the soil matrix on the dermal uptake of organic chemicals. Experimental evidence, however, suggests absorption from soil will be chemical dependent. Hence, it is important to determine dermal uptake point estimate values for specific soil-bound chemicals where appropriate data are available, as they will be more accurate than those derived on broad-based assumptions.

To obtain the ABS, a measured amount of chemical in a given amount of soil is administered to the skin surface; this amount (wt chemical/area skin) is referred to as the applied dose. The amount of chemical that crosses the skin barrier is measured and the ABS is calculated by dividing the amount absorbed by the amount applied. When measurements are made in excreta or specific organs,

corrections are included for incomplete recovery. In experiments of this type, the administered amount (in soil or solvent) represents a finite level of application. The ABS so calculated is an experimental value that is dependent upon exposure conditions, such as length of exposure and extent of soil loading. The length of exposure used for dermal exposure assessment in this document is 24 hrs. A 24 hr exposure time is commonly used in dermal absorption studies, so it's compatible for ABS calculation. In instances where absorption data did not use 24 hr exposure, an ABS will generally be based on data that are near a 24 hr exposure.

In contrast to the studies that utilize the application of finite amounts of chemicals, dermal studies that mimic scenarios such as bathing and swimming, require the applications of infinite volumes, i.e. the volume of the administered dose is much larger than the volume of the exposed skin area and the chemical at the skin surface is continuously replenished. The latter exposure scenario is not applicable to the soil studies described in this chapter, although information obtained from such studies may be useful for discussion purposes. For additional information on dermal uptake of chemicals from water (or vapor), the reader is referred to U.S. EPA (2004). The dermal absorption of chemicals from dermal exposure to contaminated water is not addressed in the "Hot Spots" program because it is likely to be a minor contribution to overall dose if it occurs at all.

F.2 Risk Assessment Issues

Although all dermal absorption studies are useful for understanding the relationship between dermal exposure and absorption, the application of these studies to risk assessment involves specific issues that must be considered to avoid development of a point estimate that may greatly underestimate, or overestimate, the potential for dermal absorption. Included among these issues are biological characteristics, soil properties, and exposure scenarios, and the variability in each can introduce uncertainties into the point estimate determination of ABS. By understanding these issues, the implications of using experimentally derived dermal ABS can be better understood. Specific categories of issues that must be considered when assessing dermal absorption are discussed below.

F.2.1 Definition of Dermal Uptake

Comprehensive dermal absorption studies often include a quantitative analysis of the amount of chemical that has passed through skin into the systemic circulation (for in vivo studies) or appears in the receptor fluid (for in vitro studies), plus the amount of chemical remaining in the skin at the site of application. Fundamentally, dermal uptake/absorption refers to the amount of dermally applied chemical that is ultimately determined to be systemically available. Because absorbed chemicals may be retained in the skin for long periods of time

and act as a reservoir for the slow systemic absorption of chemicals, the chemical remaining in skin at the end of dermal absorption experiments is considered available for systemic absorption unless data are available that shows otherwise.

Some fraction of dermally-absorbed chemicals may be only superficially diffused into skin and deposit in the stratum corneum where they are subject to counter-current forces of skin shedding, or desquamation, and ultimately removed from the body before becoming systemically absorbed. Continuous desquamation with total stratum corneum turnover has been estimated to take 2-3 weeks (Hostynek, 2003). Modeling calculations by Reddy et al. (2000) indicate that epidermal turnover can significantly reduce subsequent chemical absorption into the systemic circulation for highly lipophilic (log K_{ow} > about 4) or high molecular weight chemicals (MW > about 350-400 Da). However, some highly lipophilic chemicals retained in skin at the end of dermal absorption studies have been shown to be predominantly available for eventual absorption into the systemic circulation. Chemicals of concern that fall into this category include the PAHs and DEHP (Chu et al., 1996).

Loss of absorbed chemical through skin shedding appears to occur more readily with some hydrophilic metal salts in which a portion of the metal becomes irreversibly bound in the epidermis and subject to eventual shedding with skin. Some metal salts have such a slow diffusion (i.e., long lag time) through skin that the stratum corneum turnover rate exceeds the chemical diffusion rate (Hostynek, 2003).

Tape stripping methods to remove thin layers of stratum corneum have been used in several studies discussed below to estimate the fraction of chemical in the stratum corneum that may be lost through desquamation. A more definitive approach used in a few cases is to extend the dermal uptake study for an additional few days (after chemical is removed from skin) to determine if more of the chemical retained in the skin becomes available for systemic absorption. Other studies that help determine the fate of chemicals retained in skin include skin localization techniques and skin binding studies (Miselnicky et al., 1988; Yourick et al., 2004). But in many instances the dermal uptake studies for individual chemicals did not provide enough data to determine the fate or location of the chemical retained in skin. Thus, as discussed above, the ABS will then represent that fraction of chemical still retained in skin, plus the fraction that has already passed through the skin.

F.2.2 Dermal Bioavailability of Chemicals in Soil

The term dermal bioavailability as it applies in this section refers to the fraction of chemical in soil that is actually dermally absorbed. Dermal bioaccessibility is another term used in reference to chemical-laden soils and represents that fraction of chemical solubilized from soil, usually into water, sweat, or

gastrointestinal fluids that then becomes available for absorption. By definition, bioaccessibility should exceed bioavailability.

Published data for some chemicals considered in this section contain only data for neat application of the chemical to skin in solvent or aqueous vehicle. Generally, there is a lack of absorption data for chemicals bound to soil. To avoid potential overestimation of absorption in these instances, bioaccessibility and soil leaching studies of soil-bound chemicals are considered for adjusting the fractional absorption of the pure chemical applied to skin. These studies can be used to determine the extractable, or bioaccessible, fraction of a soil pollutant that can be deposited on the skin surface. Water added to soil is often used to determine the bioaccessibility of a soil-bound chemical, although human sweat or synthetic sweat has also been used to estimate the amount of a pollutant that can be leached from contaminated soils (Horowitz and Finley, 1993; Filon et al., 2006; Nico et al., 2006).

F.2.3 Soil - Chemical - Tissue Interaction.

Soil is a complex matrix with a highly variable composition and absorptive capacity. Organic content, mineral composition, particle size, and pH are all highly variable. Because the dermal absorption of a compound from soil is often dependent on these characteristics, it follows that transfer of a chemical from soil particles to the skin surface for absorption is likely to vary with soil type.

Transfer of a chemical from soil particles to the skin surface is limited by the chemical's diffusion rate (McKone, 1990). Diffusion through the soil phase, through the air, and through soil moisture is all possible. Fugacity-based interphase transport models were constructed to describe the rate of each of these processes for chemicals in soil particles and to predict the dermal uptake rates. It was shown that predicted dermal uptake of chemicals from soil depends on the Henry's constant (vapor pressure/solubility in water), the octanol/water partition coefficient of a chemical, and the soil thickness on skin. If the Henry's constant is very high, chemicals will be lost from soil particles (or the skin surface) quite rapidly, so net dermal uptake of chemicals added to soil will be low. If the Henry's constant is very low, diffusion through the soil particle layer will be too slow to allow much dermal uptake unless the soil particles are very small. A high octanol/water partition coefficient is associated with tight binding to soil and low water solubility; these properties also limit the ability of a chemical to diffuse through the mixed lipid/water phases of the stratum corneum.

Other mathematical models have been developed by Bunge and Parks (1997) to describe dermal absorption of organic chemicals provided the chemical fits certain assumptions, such as falling within a defined octanol/water partition coefficient range ($1.59 \leq \log_{10}K_{ow} \leq 5.53$), and that the molecular weight of the organic chemical is ≤ 700. Soil constraints for the model include contaminated soils with about 0.2% organic carbon or more, and with a clay fraction less than

60 times the weight fraction of organic carbon. The models were then used to estimate the relative effect of changing exposure conditions (e.g., changes in soil loading, contamination levels, chemical, etc.) compared to published experimental studies. Although the models were generally consistent with the experimental results for some chemicals, such as benzo(a)pyrene (BaP), they were considerably divergent from the experimental results for other chemicals, such as lindane (gamma-hexachlorocyclohexane).

The authors suggested that the fast soil release kinetics on which the models are based may not fit with what was observed experimentally for some chemicals (Bunge and Parks, 1997). Fast soil release kinetics assumes the primary resistance that controls transfer of the chemical from soil to skin resides in the dermal barrier, and that the kinetics of soil desorption are relatively insignificant. Lindane may exhibit slow soil release characteristics in various soils (i.e., soil desorption of the chemical is the controlling influence for dermal absorption), which limits the amount of dermal absorption predicted by the models.

Alternatively, Shatkin et al. (2002) developed a two-stage fugacity-based model specifically for BaP that incorporated both a fast soil desorption phase and a slow desorption phase of BaP from soil. Based on the several parameters investigated that would affect dermal bioavailability, the authors predicted that the fast desorption kinetics of a soil had a greater impact on predicted dermal uptake than any other parameter, including organic carbon content of a soil.

These examples show that the effect of soil on the dermal uptake of organic compounds can be difficult to predict without experimental data. However, dermal absorption by metal salts can be expected to be a more complex process than dermal absorption of organic compounds. Factors affecting absorption of soil-bound metals include pH, metal oxidation state, counter ion, size and solubility (Hostynek, 2003). For example, lead becomes more soluble and available for uptake in soil at low pH. However, a low soil pH tends to convert chromium (VI) to the larger less permeable chromium (III) ion. This reduction in chromium valence can also occur in transit through the skin and considerably slow the absorption of chromium through skin.

F.2.4 Effect of Soil Organic Content on Dermal Absorption

For the soil pollutants discussed in this section, one of the most common soil variables explored for effect on dermal absorption of a chemical is the organic carbon or organic matter content. The chemical adsorbed to the organic carbon phase will generally be less available for transfer to skin than neat chemical present in a separate liquid phase in the soil, largely due to strong adsorption of the chemical to the organic carbon fraction (Bunge and Parks, 1996). Dermal bioavailability of a chemical in soil also tends to decrease with increasing organic carbon content of the soil (Sheppard and Evenden, 1994; Bunge and Parks, 1997). Consequently, a number of studies compared the effect of varying the

soil organic content on the dermal absorption of a chemical. The health protective approach for estimating an ABS would be to base the value on the higher dermal absorption from these studies, often from the soil with lower organic carbon content.

The length of time required for a chemical to partition to the soil organic material may be quite short (a few days) or longer (more than a month), depending on the nature of the deposited chemical, the soil and the weather (Bunge and Parks, 1996). However, early dermal absorption studies of chemicals in soil were usually conducted with freshly spiked soil just prior to exposure. Regardless of the partitioning time to the soil organic carbon, addition of a chemical to soil can often result in a reduction of dermal bioavailability relative to the pure chemical. For a group of selected organic compounds (e.g., DDT, BaP, PCBs, etc.) and arsenic, addition to soil just before loading onto skin reduced the overall dermal uptake by an average of about 60% compared to dermal uptake of the pure chemical (Wester and Maibach, 1999). However, a reduction in absorption from soil relative to a neat solution cannot be predicted for all chemicals. Dermal absorption for some chemicals such as arsenic in soil was found to be essentially unchanged compared to absorption from the neat solution.

F.2.5 Soil Aging Effects

The ABS point estimates presented here are primarily based on soils that were freshly spiked with contaminants and placed on skin for roughly 24 hrs. As such, the ABS point estimates largely represent the initial fast phase of decreased bioavailability when a chemical is freshly added to soil prior to skin exposure (Alexander, 1995; Bunge and Parks, 1997). This phase is generally a reversible process, such that a chemical sorbed to soil may become desorbed and be available for uptake during the skin exposure.

However, over time many chemicals added to soil undergo a slower second phase of decreased bioavailability. The soil-deposited chemicals tend to move from the external surface of soil particles to internal and more remote sites within the soil matrix so that chemicals become increasingly more desorption-resistant, a process known as aging (Alexander, 1995). A number of recent dermal absorption studies discussed below have observed reductions in dermal absorption occurring for up to 3-6 months following addition of the chemical to soil. Reductions of about 50% have been observed for dermal absorption of BaP aged in soil compared to soils freshly spiked prior to skin application (Roy and Singh, 2001). Abdel-Rahman et al. (1999) observed up to a 7.5-fold reduction in dermal absorption for arsenic aged in soil.

The continuous input of chemicals deposited on soils in the vicinity of “Hot Spots” stationary sources will likely result in the less recently deposited chemicals undergoing soil aging. For toxic inorganic metals in soil, the dermal dose equation (Eq. 6.1) does not account for decreased bioaccessibility over time due

to soil ageing. Leaching and weathering effects are assumed to be very long (i.e., 10^8 days), unless site-specific information shows otherwise. Only a few studies have investigated the decrease in dermal absorption for specific inorganic metals and metalloids aged in soils, including arsenic, nickel and mercury. The soil aging results from these studies are considered in the development of the ABS, although the volume of literature available is sparse. Therefore, dermal fractional absorption still relies primarily on data for freshly applied metals to soil to avoid underestimation of the ABS.

For organic chemicals, the soil half-life variable in Eq. 6.2 will account to some degree for the effects of soil aging, depending on the rigor of the extraction process used (Abdel-Rahman et al., 2002). Use of a strong acid extraction method may solubilize some of the desorption-resistant chemical from soil and overestimate the dermal bioaccessibility of a soil-aged organic chemical. That is why milder extraction methods have been recommended, such as soil extraction in synthetic sweat, to obtain a more applicable estimate of soil half-life.

F.2.6 Dermal Soil Loading and Adherence Characteristics

The ABS from soil depends on the amount of soil in contact with the skin. Maximal fractional absorption of a soil-bound chemical occurs when a monolayer of soil covers the skin (monolayer threshold). A monolayer can be defined, in this case, as layer of soil on the skin equal in thickness to the average soil particle diameter. Theoretical calculations and experimental data show that increased soil loading (mg soil/cm^2 skin) beyond monolayer coverage usually leads to decreased fractional absorption as a result of some of the soil not being in direct contact with skin (McKone, 1990; Duff and Kissel, 1996; Bunge and Parks, 1997). Soil loading at which the monolayer exists depends on the soil particle size (Duff and Kissel, 1996). For example, sand with an average particle diameter of 0.044 cm reaches monolayer coverage at 61 mg/cm^2, whereas monolayer coverage with clay at a particle diameter of 0.0092 cm is 13 mg/cm^2 (USEPA, 2004).

Early soil loading experiments were carried out under conditions of high loading, e.g. 20-40 mg/cm^2 (Shu et al., 1988; Wester et al., 1990a; Wester et al., 1992) , without estimating monolayer coverage or providing average soil particle diameter to estimate monolayer coverage. High soil loadings that are greater than monolayer coverage may underestimate the fraction of chemical absorbed from soil. Coarse grain size (180 to 300 µm) used under the high loading conditions of 20-40 mg/cm^2 was at, or only, slightly more than monolayer coverage (Duff and Kissel, 1996). However, using such soil loadings with soils sieved to <150 µm would result in greater than monolayer coverage.

Typical soil loadings under most human exposure scenarios generally ranged from 0.01 to 0.2 mg/cm^2 when averaged over the entire exposed skin surface (USEPA, 2004). Soil loadings on the hands, the skin region with the highest soil

loading, averaged about 1 to 5 mg/cm^2 during typical human activities in wet soil with a moisture content of 9 to 18%, and usually less than 0.1 mg/cm^2 with activities in dry soil with a moisture content of 3-4% (Kissel et al., 1998).

During dermal absorption studies, the soil used to measure dermal uptake is applied to the skin as a "dry" formulation, i.e. the solvent used in the preparation of the chemical laden soil is allowed to evaporate prior to dermal application. The uptake of a soil-bound chemical from wet soil is expected to exceed the uptake from dry soil because of the increased humidity and temperature at the skin surface (Wester and Maibach, 1983). Such conditions exist for human exposure scenarios that involve high humidity, high temperature, and skin covering (e.g. gloves and clothing). Some studies are carried out under condition of occluded skin, and these studies could be used to estimate chemical absorption from soil when moisture is present.

In addition, the particle size distribution of soil adhering to skin also needs to be considered in dermal absorption studies. Most recent dermal absorption studies have sieved soil down to <150 µm prior to spiking with chemical and applying to skin. Studies have shown that soil particles in this size range tend to adhere to skin to the greatest extent (Driver et al., 1989; Sheppard and Evenden, 1994; Kissel et al., 1996). In hand press studies by Kissel et al. (1996), small particles ≤150 µm were found to adhere preferentially over larger particles ≥250 µm in dry soils of <2% moisture. Adherence in wet soils (12-18%) was roughly proportional to the soil particle size distribution of the original soil, although no consistent adherence was seen with soil moisture and particle size with five soils studied. Monolayer coverage with soil sieved to <150 µm will vary depending on the particle characteristics, but was shown in one instance to be about 2 mg/cm^2 with an estimated mean grain size of 12 µm (Duff and Kissel, 1996).

Choate et al. (2006) found that the dermally adhered fractions of two soil samples with wide distributions of particle sizes generally consisted of particles of diameters <63 µm or <125 µm, depending on the soil sampled. Adherence was similar whether the soils were applied dry (1.58-1.85% moisture) or moderately moist (3.35-3.81% moisture). With increasing moisture content of roughly 10% or greater, adherence increases significantly and a greater proportion of larger soil particles >150 µm are represented in the adhered soil (Holmes et al., 1996; Kissel et al., 1996; Choate et al., 2006). Smaller adhering soil particles can be considerably different in composition, especially in organic carbon content, from larger particles that tend to stick to skin in less abundance. However, organic carbon content does not appear to enhance the adherence of any particle sizes (Holmes et al., 1996; Choate et al., 2006).

In a few cases no dermal absorption data was available for a chemical mixed with soil. Therefore, ABS values were estimated from studies that applied the chemical directly onto the skin. Kissel (2011) observed that fractional absorption of chemicals applied neat to skin are not generally independent of skin loading

conditions. For example, the ABS will decrease as an organic chemical is increasingly loaded onto skin. In other words, absorption of an organic chemical through skin is flux-limited, and loading more chemical onto skin in a defined area will not increase flux, but will decrease the ABS value.

To aid interpretation of dermal absorption-related phenomena, Kissel (2011) proposed a dimensionless variate representing the ratio of mass delivery to plausible absorptive flux under experimental or environmental conditions. High values of this dimensionless dermal variate connote surplus supply (ie., flux-limited) conditions. This situation is similar to loading skin with chemical-bound soil above monolayer levels. The potential mismeasure of dermal absorption with chemicals applied neat to skin is addressed below for every chemical in which an ABS is derived in this way.

F.2.7 In Vivo Vs. In Vitro Experiments

It is generally recognized that the most reliable method for assessing skin absorption of a chemical is to measure penetration in vivo using the appropriate animal model or human volunteers (Kao, 1990). Thus, in vivo data are preferred over in vitro data for determination of a chemical ABS in this exposure assessment. In vivo data may be lacking for some chemicals of interest in this document due to economic~~al~~ considerations for conducting tests in humans and other mammalian species, or due to ethical concerns for testing in humans.

In vitro studies have the benefit of measuring dermal absorption under more easily controlled environments. Human skin can be tested without the inherent risks of a clinical study, and absorption through skin and retention in skin can be directly measured. Consequently, in vitro dermal absorption studies are frequently performed and provide the basis for an ABS for some chemicals presented in this section, following careful consideration for relevance to in vivo human exposure.

Although good agreement has been found when comparing in vivo and in vitro absorption results for some chemicals, trends towards lower absorption with in vitro exposure have been observed. For example, lipophilic compounds frequently have limited solubility in the buffered aqueous receptor fluids often used for in vitro cell systems, impeding the flow into the receptor fluid and resulting in an underestimation of skin penetration (Wester and Maibach, 1999). In vivo, lipophilic compounds penetrate the stratum corneum and diffuse through skin and, because of the solubilizing and emulsifying abilities of biological fluid, may readily be taken away by the blood in the dermal vasculature.

A reduction in skin viability of excised skin samples may occur due to storage conditions prior to use and may affect dermal absorption measurements. For example, the metabolic properties of human skin are reduced if the skin samples were previously frozen. Some polycyclic aromatic compounds (PAHs) undergo

extensive percutaneous metabolism when absorbed, and reducing the metabolic capabilities of skin samples will reduce dermal penetration of absorbed PAHs (Kao et al., 1985; Ng et al., 1992; Moody et al., 2009a).

For metal salts, it has been postulated that low diffusion values through the stratum corneum in vitro are a result of skin shunts (e.g., hair follicles and sweat ducts) swelling shut upon hydration of skin samples (Tregear, 1966; Hostynek, 2003). Skin shunts that bypass the stratum corneum are thought by some to be a significant absorption route for charged metals. For example, dermal absorption of nickel salts shows there is a surge in diffusion at the earliest stage, which then rapidly decreases towards steady state (Tanojo et al., 2001). The decrease in diffusion rate has been proposed to be a result of the skin tissue becoming hydrated, shutting down the skin shunts.

A further potential limitation under in vitro conditions is that diffusing compounds must traverse the epidermis and the entire dermis in order to reach the receptor fluid. In vivo, the majority of the absorption into the cutaneous microcirculation is thought to occur in the upper dermis and the penetrant compounds may not have to diffuse across the entire thickness of the dermis. However, the bulk of the connective tissue in the dermis is often eliminated from the skin preparation by cutting the skin parallel to the skin surface with a dermatome (Poet and McDougal, 2002).

In vivo studies are not without limitations. Dermally applied chemicals are often radiolabeled to facilitate quantification of the usually low absolute amounts of chemical dermally absorbed. In small mammals, a total accounting of all dermally absorbed radioactivity can be estimated from excreta, carcass, and site of skin absorption. However, in larger mammals measurements of radiotracer are quantified in excreta and measurements from intravenous, intramuscular, or oral dosing are applied as a correction for tissue absorbed chemical. The validity of this method depends on the underlying assumption that metabolism and disposition of the applied compound is route independent, and that the pharmacokinetic behavior of the intravenous and topical doses is similar (Kao, 1990).

F.2.8 Inter- and Intra-Species Specificity

The variability in dermal absorption of chemicals among mammalian species has been investigated in vivo and in vitro. Bartek et al. (1972) suggest that the extent of in vivo uptake among animals follows the rank: rabbit > rat > pig ≈ monkey ≈ humans, based on dermal absorption of benzoic acid, hydrocortisone, testosterone, caffeine, N-acetylcysteine, and butter yellow. However, the species ranking did not strictly hold for all chemicals, indicating not only species-specific differences but also chemical-specific differences.

Comparison of data from other studies does support that in general, the absorption in the rabbit, rat and other rodents can considerably overestimate absorption in humans, while absorption in monkeys and miniature pigs most closely predict human absorption (Wester and Maibach, 1975; Reifenrath et al., 1984; Wester and Maibach, 1985; Bronaugh et al., 1990; Wester et al., 1998a). Alternatively, Kao et al. (1985) found that in vitro permeation of testosterone and BaP through human skin was greater than that for guinea pig, rat, or rabbit, indicating that species-specificity differences likely depend on other factors such as experimental conditions and tissue viability. Variability in dermal absorption depending on the skin area exposed has been investigated (Wester and Maibach, 1983). In humans, absorption across the skin varies by area of the body and may be higher than the commonly used forearm (e.g. scalp, axilla, forehead, jaw angle and scrotum).

F.2.9 Metabolism of Absorbed Chemicals in the Skin

The description of percutaneous absorption is generally based on diffusion models that take into account the physico-chemical characteristics of chemicals and soils. While such descriptions may help to explain the uptake of chemicals across the stratum corneum, the role played by metabolism in the viable epidermal and dermal layers should be included to understand the complete permeation of chemicals through the skin (Wester and Maibach, 1983; Kao and Carver, 1990; Bronaugh et al., 1994).

Viability of the skin refers to the status of active energy turnover, i.e. the utilization of glucose and formation of CO_2 or lactate in skin. Enzymes and metabolic processes in skin may affect the dermal penetration of drugs and other xenobiotics, particularly if absorbed chemicals can be metabolized in the skin. Using production of lactose as the measure of viability, human skin placed in a buffered solution and kept refrigerated remained viable for about 8 days following donor death (Wester et al., 1998b). Skin frozen for storage or heat-treated to separate the epidermis and dermis renders the skin non-viable and may change the dermal penetration dynamics of absorbed chemicals. Some early studies investigating the dermal penetration of chemicals used previously frozen skin samples and may not provide a good basis for ABS determination.

Dermal metabolism of BaP was observed to be considerably reduced in several mammalian species with use of non-viable skin, resulting in reduced penetration of BaP through skin (Kao et al., 1985). In viable human skin, nearly half the BaP that permeated the skin was attributed to BaP metabolites. In non-viable skin, essentially only unchanged BaP was detected in the receptor fluid. In fact, dermal absorption of polycyclic aromatic hydrocarbons (PAH) that include BaP resulted in PAH-DNA adducts in human skin samples, demonstrating that skin is a target organ due to metabolic activation of PAHs in skin (Phillips et al., 1990).

On the other hand, dermal absorption of some chemicals does not appear to be affected by the viability status of the skin samples. Dermal penetration of TCDD through viable and non-viable pig skin was found to be similar (Weber, 1993).

F.2.10 Human Adult and Infant Variability in Skin Permeability

Animal studies are designed to ensure uniformity within the experimental population by using inbred strains and often only one sex. The variability between animals is much less than the genetically diverse human population. Human studies also rarely use children or infants, the elderly, pregnant women and the infirm, partially because of ethical considerations. Dermal uptake may vary due to genetic diversity in the human population and differences in age. This variability will not necessarily be accounted for by experimental data.

A review of the data on human skin permeability to chemicals suggest at least a mean intra-individual coefficient of variation of approximately 40% and a mean inter-individual variation of about 70% (Loth et al., 2000; Hostynek, 2003). A leading cause in the variation is the lipid composition of the stratum corneum, which influences solubility and permeability of drugs. This factor is partly responsible for the high variability in accumulation and permeation measurements (Loth et al., 2000).

There has been increasing awareness in recent years that infants and children are more susceptible than adults to the harmful effects of some pollutants. This can be due to differences in exposure, physiology, absorption, distribution, metabolism, and excretion. Further, organ development and faster cell division influence targets of toxicity. Finally, a large skin surface area to body weight ratio would increase the dose of an absorbed chemical on a mg/kg body weight basis.

Only a few studies have examined age-related differences in the dermal absorption capacity of chemicals in infants and children compared to adults. Preterm infants lack a fully developed dermal barrier function and are particularly prone to accidental poisoning of toxic agents applied to the skin surface (Barrett and Rutter, 1994). In an in vitro system, McCormack et al. (1982) observed increased penetration of some alcohols and fatty acids through skin of premature infants compared to full term infant skin and adult skin. Dermal absorption of sodium salicylate was found to be a hundred- to a thousand-fold greater in infants of 30 weeks gestation or less compared to full term infants (Barker et al., 1987).

In full-term infants, epidermal structure and function matures by 2-3 weeks of age (Holbrook, 1998; Makri et al., 2004). In general, the in vitro system of McCormack et al. (1982) showed full-term baby skin to be a good barrier for some compounds. No difference in penetration of alcohols through full term infant and adult skin was seen. However, penetration of some fatty acids through full term infant skin was greater than that through adult skin. Higher lipid

content in the stratum corneum of infants was thought to be the reason for increased absorption of fatty acids. In addition, a layer of subcutaneous fat develops at approximately 2-3 months of age in infants and continues to exist through the early toddler period (Thompson, 1946; Banks et al., 1990; Cohen Hubal et al., 2000). This layer of fat may act as a sink for lipophilic chemicals absorbed through the skin.

Age-related changes in dermal absorption have also been investigated in experimental animal models. Using TCDD or 2,3,4,7,8-pentachlorodibenzo-p-dioxin (4-PeCDD) in solvent, Banks et al. (1990) observed greater absorption of TCDD or 4-PeCDD in 10-week old rats than 36 - 120-week old rats. 2,4,5,2',4',5'-Hexachlorobiphenyl showed significantly higher fractional penetration in young rats (33 days old) compared to adult rats (82 days old) in vivo, but only at one of three dose levels tested (Shah et al., 1987). Overall, the authors concluded that no clear age-related pattern of dermal absorption was found among a total of 14 pesticides including 2,4,5,2',4',5'-hexachlorobiphenyl.

F.2.11 Use of Default ABS Values

The California South Coast Air Quality Management District's Multi-Pathway Health Risk Assessment Input Parameters Guidance Document (SCAQMD, 1988) recommended using default values of 10% for organic chemicals and 1% for inorganic chemicals when quantitative data are not available to estimate chemical-specific dermal absorption fractions from soil.

Use of these default factors was proposed based on a review of the dermal absorption literature and recommendations by McLaughlin (1984). In his US EPA report, McLaughlin suggests it may be possible to group penetrants into a numerical system using an "order of magnitude" approach (i.e., 100% - 10% - 1% - 0.1% fractional absorption groupings), depending on physical parameters such as partition coefficients and diffusion constants. For example, many of the organic compounds were found to fall into the 10% absorption range. Exceptions included some pesticides, such as the very lipophilic pesticide carbaryl that exhibited a fractional absorption closer to 100%, and the polar pesticide diquat that exhibited a fractional absorption closer to 1%.

More recently, US EPA (2004) also recommended a default dermal absorption fraction for semivolatile organic compounds (SVOCs) of 10% as a screening method for the majority of SVOCs without dermal absorption values. This fraction was suggested because the experimental values for SVOCs determined by US EPA are assumed to be representative of all SVOCs as a class. US EPA (2004) notes that chemicals within classes can vary widely in structure and chemical properties, potentially resulting in a wide range of fractional absorption values. However, OEHHA agrees that a 10% fractional absorption default value is acceptable at this time, based on the range of values (3 to 14%) estimated in

Table F.5 for SVOCs. Currently, the OEHHA default ABS value for organic compounds applies only to 4,4'-methylene dianiline.

For inorganic classes of compounds, US EPA (2004) recommended that no default dermal absorption values be used. The premise was that speciation of inorganic compounds is critical to the dermal absorption and there are too little data to extrapolate a reasonable default value. With the exception of the metalloid arsenic, OEHHA notes that the range of ABS point estimate values for inorganic the metal and semi-metal salts (see Table F.5) is relatively narrow, between 0.2 and 46%. Therefore, it is reasonable to assume that a default ABS of 13% can be used as a screening value, primarily if there are some data to indicate that the metal salt exhibits characteristics of low fractional dermal uptake similar to other metal saltsbased on the mean ABS value for the metals and semi-metals in which published dermal absorption data exists (i.e., arsenic, cadmium, hexavalent chromium, lead, mercury and nickel). Currently, OEHHA the default ABS value for inorganic compounds applies only to inorganic compounds of fluoride, beryllium and selenium.

F. 3 Point Estimates for Dermal Absorption (ABS) of Inorganic Compounds

F. 3.1 Arsenic and Arsenic Compounds

Recommended point estimate for dermal uptake: 6%.

F.3.1.1 Studies Considered

A. Key Studies

Wester et al. (1993a) examined the in vivo percutaneous absorption of radiolabeled soluble arsenic (as $H_3{}^{73}AsO_4$) freshly mixed with soil and applied to skin of female Rhesus monkeys (n = 4 animals per dose group). Dose levels of 0.0004 and 0.6 μg/cm^2 were used. The soil load on the skin was 40 mg soil/cm^2 skin area. The soil had been sieved to 180-300 μm prior to application, thus, a soil load of 40 mg/cm^2 was likely at or near monolayer coverage. Topical doses were applied to an area of the abdomen for 24 hours. Urine was collected during the dosing period, and through the following 6 days. For comparison, radiolabeled arsenic (as ^{73}As) in water was administered intravenously to four monkeys. Percutaneous absorption was determined by the ratio of urinary arsenic excretion following topical application to that following intravenous administration.

Urinary excretion of the ^{73}As label was complete by day 7, with about half the label excreted in the first 24-48 hrs following topical administration. Results of this study showed that the percutaneous absorption of arsenic from soil was 4.5

± 3.2% from the low dose and 3.2 ± 1.9% from the high dose (nonsignificant difference). An estimate of arsenic retained in the skin was not performed, although 27-28% of the arsenic could not be accounted for following decontamination of the skin.

Lowney et al., (2005) conducted follow-up absorption studies with arsenic aged in soil that paralleled the methodology used in the in vivo Rhesus monkey study. The soil samples collected were adjacent to a pesticide production facility that had historically produced calcium and lead arsenate compounds. The arsenic was resident in the soil for a minimum of 30 years and was primarily in the sparingly soluble iron oxide and iron silicate mineral phases. Small amounts of more soluble calcium arsenate and arsenic trioxide were also detected in the soil. The particle size fraction was sieved to <150 µm and a skin loading of 4 mg/cm^2 on 100 cm^2 of skin was applied. Total dose was 560 µg arsenic and the duration of dermal exposure was 8 hrs on the abdomens of three monkeys. Following fractional correction of arsenic from i.v. dose, urinary excretion of arsenic ranged from 0.01 to 0.24% of the dermally applied dose, but was not statistically greater than background. Negligible absorption was considered due to the presence of soil arsenic primarily in sparingly soluble mineral phases. Direct or indirect estimates of arsenic retained in the skin were not performed.

A sweat extraction technique by Nico et al. (2006) was employed to estimate the soluble arsenic that can be made bioavailable for dermal absorption from the aged arsenic soil used in the in vivo monkey study by Lowney et al. (2005). Sweat extraction of this soil resulted in only 1.8% soluble arsenic. However, a second aged soil sample from a different arsenic-contaminated site resulted in 11% arsenic extracted by sweat. Nico et al. (2006) also used the sweat extraction technique to estimate soluble arsenic from soil samples freshly spiked with arsenic. One sample was sieved to <150 µm while another was sieved to 180-300 µm, similar to that used by Wester et al. (1993a) in the in vivo dermal monkey study. Sweat extraction resulted in 45 and 72% soluble arsenic from the <150 and 180-300 µm soil samples, respectively.

B. Supporting Studies

In addition to the monkey in vivo study, Wester et al., (1993a) conducted an in vitro study using human cadaver skin from three separate donor sources with three replicates from each source. The skin was dermatomed to 500 µm, stored refrigerated in Eagle's medium and used within 5 days to preserve skin viability, although elapsed time from death to harvest of skin was not specified. A dose of 0.0004 µg arsenic per cm^2 skin surface exposed was applied. The soil load on the skin samples was 40 mg soil per cm^2 skin area, and phosphate-buffered saline served as receptor fluid. The in vitro exposure period was 24 hours. As performed in the monkey in vivo study, the soil had been sieved to 180-300 µm prior to application, so monolayer coverage was probably not surpassed. Percutaneous absorption through human cadaver skin was 0.76% (0.43% in

receptor fluid; 0.33% in skin) after soap and water wash. While the authors did not speculate as to the reduced in vitro dermal absorption compared to monkey in vivo absorption, Kao (1990) noted that both elapsed time from death to harvest of tissues and treatments and storage of the cadaver could have resulted in a large variability in skin permeability.

Dermal absorption of radiolabeled soluble arsenic (as $H_3{}^{73}AsO_4$) freshly applied or aged in two different soils was determined in vitro through dermatomed pig skin cut 200 µm thick (Abdel-Rahman et al., 1996; Abdel-Rahman et al., 1999). Soil types included a sandy soil with 4.4% organic matter and a clay soil with 1.6% organic matter, with no apparent sieving before application. Arsenic was applied to skin for 16 hrs either alone in ethanol vehicle, immediately after the addition of 30 mg of the soils to skin, or after aging for 3 months in each soil. Soil loading was calculated to be about 47 mg/cm^2. Applying soil to skin and then applying the arsenic does not allow time for arsenic-soil equilibrium. This method of application allows for direct contact of skin with arsenic or vehicle and not from soil, leading to an overestimation of the fractional absorption (Spalt et al., 2009). In addition, monolayer coverage was probably exceeded with a soil loading of 47 mg/cm^2.

With arsenic freshly added to soil, 0.2% of the arsenic penetrated the skin to receptor fluid from both soil types (Abdel-Rahman et al., 1996; Abdel-Rahman et al., 1999). Total dermal absorption including arsenic retained in skin was 10.0 and 6.0% from the sandy and clay soils, respectively. In comparison, pure arsenic found in receptor fluid and retained in skin was 0.4 and 44.2%, respectively. In aged sandy and clay soil, 0.2 and 0.1% arsenic was found in the receptor fluid, respectively. Total dermal absorption in the aged soils was 1.5 and 0.8% from sandy and clay soils, respectively.

Radiolabeled sodium arsenate was applied in vitro to the skin of mice for 24 hrs as a solid compound, in an aqueous solution, or as an aqueous solution in sandy soil (Rahman et al., 1994). Soil was sieved to $\leq$180 µm and contained 58% sand, 34% silt, 8% clay and 1.4% organic matter. Arsenate was freshly applied to soil prior to skin application, with an average soil loading on the skin of 23 mg/cm^2. Absorption increased linearly with the applied dose from all exposure vehicles, with a constant fraction of the dose being absorbed. Total arsenate absorption was as high as 62% of applied dose from 100 µl water vehicle and about 33% of applied dose as the solid. However, absorption of arsenate from soil was less than 0.3% of applied dose, with about one-third penetrating to the receptor fluid.

A dermal exposure study was conducted to assess the potential for arsenic exposure in children in contact with playground equipment and decks treated with the wood preservative chromated copper-arsenate (CCA) (Wester et al., 2004). Methodology was similar to that used by Wester et al. (1993a) in three monkeys to assess dermal arsenic absorption from CCA-treated wood residues.

Following 8-hr dermal application, an increase in urinary excretion of arsenic above background was not detectable, indicating virtually no absorption of arsenic from CCA-treated wood residue. The researchers determined that the absorbed dose would need to be in the range of 0.10 to 0.16% of the applied dose to be detectable above background.

The negligible dermal absorption of arsenic from the CCA residues is a result of arsenic chemically bound with other metals (particularly chromium) and ultimately to the wood structure (Nico et al., 2004). The leaching characteristics of soluble arsenic in CCA residues were also investigated by extraction in human sweat (Nico et al., 2006). The sweat extraction procedure indicated that up to 12% of total arsenic is available for dermal absorption from CCA-treated wood residue. However, only 1.4% soluble arsenic was extracted with sweat from CCA-residue aged in soil near a CCA-treated utility pole. Gastric leaching conditions resulted in up to 2-3 times greater solubilization of arsenic from CCA-treated wood compared to sweat leaching, indicating soil ingestion of CCA-released arsenic can be a health concern.

F. 3.1.2 Discussion and Recommendation for Arsenic and Arsenic Compounds ABS

Dermal exposure of skin to arsenic resulting in passage of arsenic through skin to the bloodstream is the primary concern under the “Hot Spots” program. However, arsenic that becomes bound in skin may also have toxicological consequences. Regardless of route of exposure to arsenic the skin is a critical target organ for arsenic toxicity due to local absorption and binding of sulfhydryl-group-containing proteins (Hostynek et al., 1993). The affinity for sulfhydryl groups leads to arsenic’s accumulation and tenacious retention in keratin-rich tissues such as hair, nails, and skin. Measurement of in vitro percutaneous absorption of As(III) and As(V) by human epidermal skin cultures for 6 hrs shows strong affinity of arsenic for the keratinocytes, with an estimated 30% of As(V) passing through skin being retained compared to over 90% of the As(III) being retained (Bernstam et al., 2002).

Accumulation of arsenic in the skin is characterized by hyperpigmentation, keratoses of the palms of the hands and soles of the feet, and diffuse macular pigmentation or diffuse darkening of the skin on the limbs and trunk, attributed to the reduction and deposition of the element in the metallic state (Hostynek, 2003). Chronic arsenic accumulation in skin increases the susceptibility of the skin to ultraviolet light and is associated with an increased incidence of tumors of exposed skin, although skin cancer is primarily a result of oral arsenical poisoning and characterized by multifocal lesions over the entire body (Hostynek et al., 1993; OEHHA, 1999).

The key in vivo monkey study by Wester et al. (1993a) provides an average fractional absorption of 3.9% based on two dose levels of arsenic that had been

freshly added to soil before application to skin. Some limitations are noted for this study. First, the in vivo study did not estimate arsenic retained in skin. However, the researchers followed excretion of arsenic after exposure and noted that excretion of the labeled arsenic was essentially over by day 7. The remaining arsenic bound to skin proteins will probably remain there and not present a risk of reaching the bloodstream.

Secondly, a sieved soil fraction of 180-300 µm was used, which does not reflect the generally smaller soil particle fraction that sticks to skin following dermal contact. Soil sieved to <150 µm is considered more relevant for dermal studies (Spalt et al., 2009). The sieved soil used by Wester et al. may underestimate fractional absorption. This assumption is supported by the sweat extraction study by Nico et al. (2006), which found a 63% increase in arsenic bioavailability (45% to 72%) from soil sieved to <150 µm as opposed soil sieved to 180-300 µm.

Finally, there is also some question whether the contaminated soil had continuous contact with the skin of the monkeys (Spalt et al., 2009). From the methodology description, the eye patches used to hold the soil in place on the abdomen of the monkeys were a larger volume than the applied soil. Thus, sloughing of soil off the skin probably occurred when the monkeys sat upright.

Together, these limitations indicate that basing an ABS on the monkey study may underestimate the dermal fractional absorption of arsenic. However, the sweat extraction study by Nico et al. (2006) supports the application of an adjustment to account for use of a soil fraction that likely underestimates fractional absorption. A 63% increase in arsenic bioavailability was observed from soil sieved to <150 µm, compared to soil sieved to 180-300 µm, as used by Wester et al. (1993a). A soil sieved to <150 µm better characterizes the soil particle size that adheres to skin. Thus, a 63% increase was applied to the monkey fraction absorption value of 3.9% resulting in an arsenic ABS of 6% when rounded to the nearest whole number.

The in vitro studies reviewed here gave a range of 0.3 to 10% for total absorption following application of freshly spiked soil to skin samples (Rahman et al., 1994; Abdel-Rahman et al., 1996; Abdel-Rahman et al., 1999; Wester et al., 1993a). However, arsenic aged in two soils gave a total dermal absorption of 0.8-1.5% in pig skin in vitro (Abdel-Rahman et al., 1996). As discussed above, it is difficult to reconcile the difference in dermal absorption in pig skin between arsenic freshly spiked in soil and arsenic aged soil due to differences in methodology. Future in vitro studies using human skin and arsenic freshly applied and aged in soils would help assess the impact of arsenic aged in soil.

F. 3.2 Beryllium and Beryllium Compounds

Recommended use of default inorganic compound ABS estimate of ~~1~~3.0%.

F. 3.2.1 Studies Considered

No quantitative data could be found regarding the fractional dermal absorption or skin penetration of beryllium (Be) compounds. Be metal powder can oxidize when suspended in synthetic sweat, whereupon the metallic ions may be absorbed in human skin (Larese et al., 2007). However, Be salts are corrosive to skin, and have a high reactivity with protein substrates that result in strong retention in skin (Hostynek et al., 1993). The reaction of beryllium salts with the proteins in skin acts as a strong sensitizer that cause allergic contact dermatitis. Beryllium compounds typically decompose to form the poorly soluble, amorphous oxide (BeO) or hydroxide ($Be(OH)_2$), resulting in tissue granulomas (i.e., compactly grouped cells that replace normally functioning tissue) and ulcers. Once lodged in tissue, these amorphous beryllium precipitates are excreted at a very slow rate.

Belman (1969) investigated the interaction of beryllium fluoride and beryllium sulfate with guinea pig epidermal tissue in order to explore a mechanism for the delayed allergic skin reaction observed in humans following beryllium exposure. Using both in vitro and in vivo experiments, he reported that beryllium is taken up into the skin and localized primarily to proteins of the epidermis, with little or no apparent binding to stratum corneum or dermis. Exposure caused a localized immune response and rapid destruction of skin cells. Data are not provided, however, regarding the amount of beryllium taken up by the skin cells, or the fate of beryllium following the immunological response (i.e., whether beryllium is then absorbed into the circulation, or sloughed off with cells.)

Petzow and Zorn (1974) reported on the absorption of beryllium through the tail skin of rats exposed to an aqueous beryllium chloride solution spiked with ^{7}Be. The authors stated that within the first hour of exposure there is an increase in the rate of beryllium uptake. After approximately 90 minutes, the dermal flux of beryllium from the aqueous solution is constant. In addition, Petzow and Zorn reported that the amount of beryllium that diffuses through the skin seems to be dependent upon the concentration of beryllium in contact with the skin.

Worker exposure and likely facility emissions of beryllium compounds are mostly in the form of particulates, primarily BeO (Tinkle et al., 2003; Day et al., 2006). For these poorly soluble beryllium particles, dermal exposure is considered to be of toxicological significance. Chronic beryllium disease (CBD) is an occupational disease that begins as a cell-mediated immune response to inhaled beryllium. Although respiratory and engineering controls have significantly decreased occupational inhalation exposures, reduction in occurrence of beryllium sensitization and CBD has not significantly decreased. The lack of worker skin protection has been postulated as a contributor to the persistence of sensitization and CBD in the workplace.

The concentration of antigen required for elicitation of a cell-mediated immune response is significantly smaller than the concentration required for sensitization, therefore, the failure of respiratory exposure limits to lower the rate of disease is likely related to the continued unchecked skin exposure to beryllium particles (Tinkle et al., 2003; Day et al., 2006; Deubner and Kent, 2007). Thus, in workers with significant beryllium skin exposure, the pulmonary exposure required to elicit a subsequent immune response and granuloma formation would be significantly smaller.

To determine if BeO can penetrate the stratum corneum and reach the immunologically active epidermis, Tinkle et al. (2003) conducted a pilot study in which BeO particles were suspended in petrolatum (1 mg/g), painted on the back of shaved mice, and the area covered with surgical tape. The average amount of beryllium applied to each mouse was 70 µg. Excess BeO was removed from the surface of the flank skin by gentle washing and tape stripping three times immediately following 24-hr exposure. On day 7 or 14 following the exposure, the amount of beryllium in the flank skin of BeO-treated mice was, on average, 1.2 µg/g tissue, thus confirming that BeO is present in the skin.

Additionally, Tinkle et al. (2003) observed in vitro that polystyrene latex spheres <1 µm in diameter, when applied to skin and coupled with flexing motion, can penetrate intact human skin. The researchers proposed that beryllium particles can similarly penetrate the skin.

F. 3.2.2 Discussion and Recommendation for the Beryllium and Beryllium Compound ABS

Due to the lack of quantitative data regarding dermal absorption of beryllium, it is not possible to calculate a chemical-specific fractional absorption value for Be salts. The high reactivity of beryllium with skin suggests penetration to the bloodstream in intact skin is small relative to other inorganic metals discussed in this section. However, it is postulated that a primary concern for dermal exposure to beryllium is related to sensitization, which results in much lower inhaled concentrations of beryllium particles required for elicitation of a cell-mediated immune response leading to progression of CBD (Tinkle et al., 2003; Day et al., 2006). This action only requires penetration to the epidermis where the immune response occurs. Considering that full dermal penetration of beryllium to the bloodstream may not be required to enhance or facilitate a toxicological response, and that particles have been shown to penetrate the skin with flexing, it is recommended that an ABS of 3, based on the mean ABS for the other Hot Spot metals (Cd, Cr(VI), Pb, Hg, Ni) and semi-metals (As), be used for beryllium ~~a default ABS of 1% for inorganic beryllium compounds in soil be used~~ for screening purposes to assess dermal exposure.

F. 3.3 Cadmium and Cadmium Compounds

Recommended point estimate for dermal uptake: 0.2%.

F. 3.3.1 Studies Considered

A. Key Studies

Wester et al. (1992) examined the percutaneous absorption of cadmium chloride from soil using human cadaver skin in an in vitro system. Donor skin was used within 5 days of harvest and was kept refrigerated in buffered medium until then. The soil used prior to sieving contained 26% sand, 26% clay, 48% silt and 0.9% organic carbon. The soil was sieved to retain particles in the range of 180 to 300 µm. Radiolabeled cadmium (^{109}Cd) was mixed with soil at a concentration of 13 ppb and applied to the skin samples at a soil loading of 20 mg/cm^2 or 40 mg/cm^2. Two donor skin sources were used with replicates for each of the soil concentrations. Human plasma was used as the receptor fluid. At the end of a 16-hour exposure, soil was removed from the samples by soap and water rinse. Percutaneous absorption, calculated as receptor fluid accumulation plus residual skin concentration after soap and water wash, ranged from 0.08% to 0.2% of applied dose (Table F.1). No significant differences were observed in absorption between skin samples or soil load concentrations.

Table F.1. In Vitro Human Dermal Fractional Absorption of Cadmium Chloride from Soil[a]

Soil Loading	Skin Source	Percentage Applied Dose		
		Receptor Fluid	Skin	Total
40 mg/cm^2	1	0.02 ± 0.01	0.06 ± 0.02	0.08
	2	0.07 ± 0.03	0.13 ± 0.05	0.20
20 mg/cm^2	3	0.02 ± 0.02	0.08 ± 0.06	0.1
	4	0.02 ± 0.02	0.08 ± 0.06	0.1

[a] Data from Wester et al. (1992); n = 3 replicates per skin source

In another experiment, Wester et al. (1992) applied cadmium in water to human skin samples for 30 min, followed by removal of the cadmium solution from the skin surface and continued perfusion of the skin for an additional 48 hrs. No cadmium appeared in the receptor fluid after 30 min of exposure. However, 0.6 ± 0.8% of the dose had diffused into the receptor fluid after 48 hrs demonstrating the capacity of cadmium to be retained in the skin and be slowly systemically absorbed over time.

B. Supporting Studies

Kimura and Otaki (1972) used liver and kidney accumulation of cadmium in rabbits and hairless mice to estimate dermal absorption. A total dose of 30.5 mg Cd (in an aqueous $CdCl_2$ solution) was administered to rabbit skin (n=1) in 5 doses over 3 weeks. Two weeks after the final application, 0.40% of the applied dose was found in liver and kidney combined. In rabbits (n=2), a total dose of 61 mg Cd was administered in multiple cream-like and milk-like ointment applications, resulting in 0.45 and 0.61%of the applied dose, respectively, in liver and kidney combined. The type of ointment vehicle used did not appear to greatly affect the absorption or accumulation characteristics of Cd. Dermal absorption of cadmium in hairless mice, estimated from kidney and liver accumulation, ranged from 0.07-0.27% after a single application of ointment (0.61 mg Cd). Cadmium absorption after multiple ointment applications on hairless mice ranged from 0.59 - 0.87% of applied dose.

Aqueous 1.0, 0.1 and 0.01% cadmium solutions were painted onto the skin of mice and rats and air dried each day for ten days (Lansdown and Sampson, 1996). Perceptible skin damage occurred at the two highest doses, likely resulting in increased dermal absorption. At the lowest dose, significantly increased skin content of cadmium was observed in both mice (138 ng Cd/g) and rats (248 ng Cd/g). Adequate data to estimate fractional absorption were not provided.

Although no studies estimated dermal absorption of cadmium aged in soils, Aringhieri et al. (1985) reported that 80% of cadmium added to a soil containing high organic matter (14.2%) and high clay content (60%) was adsorbed to soil particles within 10 min of addition to a soil. Tang et al. (2006) observed that bioaccessibility of cadmium (relating closely to absorption following ingestion of soil) in strongly acidic soils spiked with cadmium reached nearly steady state levels as high as 77% after the first week of aging. In soils highly contaminated with heavy metals by industrial sources, the $MgCl_2$-exchangeable fraction of cadmium was about 37% and was considered the most mobile and biologically available heavy metal in the samples examined (Hickey and Kittrick, 1984).

F. 3.3.2 Discussion and Recommendation for a Cadmium and Cadmium Compounds ABS

No in vivo studies investigating fractional absorption of cadmium from soil were located. The human in vitro study by Wester et al. (1992) provided the only quantitative data for dermal absorption of cadmium from soil. The retention and concentrating of cadmium in skin with slow systemic absorption demonstrate the necessity for including the cadmium found in exposed skin for estimating an ABS point estimate.

The lack of quantitative in vivo studies and the use of 16 hr rather than 24 hr exposures support a point estimate based on the highest fractional absorption of 0.2%, rather than a the lower estimate of 0.1% (based on an averaging of

different skin sources for each of the two soil loadings). In addition, coarse particle soil loadings of 20 and 40 mg/cm^2 may result in a reduced fractional absorption, although the data suggest monolayer coverage of skin was probably not exceeded (Spalt et al., 2009). The high bioavailability and apparent low capacity for aging of cadmium in some soils indicates that sequestration of cadmium in soil will be small relative to other inorganic metals in soil.

F. 3.4 Soluble Compounds of Hexavalent Chromium

Recommended point estimate for dermal uptake: 2%

F. 3.4.1 Studies Considered

A. Key Study

Czernielewski et al. (1965) exposed guinea pigs to hexavalent chromium (chromium (VI)) as sodium chromate solution labeled with Cr^{51}. A single dose (15 μg sodium chromate in 0.1 ml solution) was applied to a 4 cm^2 shaved area of skin for 24 hours (n=9 animals). Absorption was estimated by measurement of the Cr^{51} content of the following: urine, feces, blood (1 ml), heart, liver, spleen, adrenals, kidneys, lungs, lymphatics, and skin. Dermal absorption of chromium (VI) was estimated to be 2.9% of the applied dose from the 24 hour exposure. Based on the average blood volume of adult guinea pigs (27 ml), 1.6% of applied dose was found in blood, 1.1% in excreta, and only 0.2% in organs and tissues including skin.

B. Supporting Studies

Chromium in the hexavalent [Cr(VI)] state does not measurably bind with proteins, whereas the trivalent chromic ion [Cr(III)] shows strong affinity for protein in epithelial and dermal tissues (Samitz et al., 1969; Gammelgaard et al., 1992). Thus, Cr(VI) can permeate through skin relatively easily compared to Cr(III). However, skin has the capacity, though limited, to reduce Cr(VI) to Cr(III) resulting in binding of chromium to skin protein and decreasing the rate of diffusion (Gammelgaard et al., 1992; Hostynek, 2003). Binding of chromium in the skin is characterized as irreversible, leading to protein denaturation with formation of permanent depots in the epidermis (Hostynek, 2003). Some of the bound chromium is likely subject to the counter-current effect of continuous sloughing of the outer skin layers, although no studies have attempted to quantify this removal pathway.

To investigate the level of penetration of Cr(VI) into human skin, Liden and Lundberg (1979) cut 10 μm tangential sections of skin biopsies after application of a 0.5% aqueous potassium chromate solution on a 79 mm^2 patch of skin on the back of volunteers. Dermal exposure durations to the chromate were 5, 24, or 72 hrs. Highest chromium levels were found in stratum corneum. Chromium

was also found at the dermal-epidermal junction and the upper mid-dermis. Chromium levels differed considerably between different biopsies, but the content of chromium was the same order of magnitude at all exposure durations indicating that a steady state was reached within 5 hrs of exposure.

Alternatively, Mali et al. (1964) measured the disappearance of a radiolabeled chromate solution absorbed dermally in two human volunteers and determined penetration into stratum corneum by tape stripping. Application of a 0.02 ml 0.25% dichromate solution (containing 50 µg Cr(VI)) on a patch to the arm for 12 hrs resulted in the disappearance, and presumed absorption, of 22 µg Cr into the skin. Tape stripping of stratum corneum removed 0.35 µg of radiolabel in the skin.

Systemic uptake of chromium was studied in four human volunteers following a three hour submersion in a tub of water containing 22 mg/L Cr(VI) as potassium dichromate (Corbett et al., 1997). Urinary chromium excretion showed large intra-individual variability. Five-day total Cr urinary excretion above historical background ranged from 17.5 to 1.4 µg, with an average of 6.1 µg. Urine levels of chromium were normal in three volunteers by day 2, although a fourth volunteer excreted elevated levels of chromium up to the end of the experiment on day 5. Elevated blood and serum levels of chromium were recorded within 1 hr after end of exposure. Chromium content of red blood cells was generally increased about 2-fold, and serum content was increased about 3- to 5-fold. Chromium levels in red blood cells and serum had returned to control levels 2 days after exposure. The systemic uptake rate through skin ranged from 4.1E-04 to 7.5E-05 µg/cm^2-hr with an average of 1.5E-04 µg/cm^2-hr.

Aqueous solutions of Cr(VI) as potassium dichromate, and Cr(III) as chromium trichloride and chromium nitrate were applied in vitro to full thickness human abdominal skin in diffusion cells at a chromium content of 0.034 M (Gammelgaard et al., 1992). Test solutions of 556 µl/cm^2 were applied over a skin surface area of 1.8 or 0.7 cm^2. After 190 hrs exposure of skin to the dichromate, 134 and 12 µg Cr/cm^2 were found in the epidermis and dermis, respectively. Only 0.037 µg Cr/cm^2 was found in the recipient phase. A total Cr(VI) permeation of 15% was calculated. Significantly less Cr(III) from either the trichloride or nitrate was found in skin. Cr(III) content in skin was no more than 9% of the chromium content applied as Cr(VI), with no chromium found in the recipient phase. The lower permeation of Cr(III) was considered a result of the skin acting as a barrier to absorption of the positive Cr(III) ions.

In other experiments by Gammelgaard et al. (1992), application of the dichromate at concentrations of 0.125, 0.25, and 0.5% to skin for 48 hrs showed increased Cr content in skin with increasing concentration, although no Cr was detected in the recipient phase. Total percent Cr permeation of 0.7, 0.7 and 1.1% was calculated for exposure to the 0.5, 0.25 and 0.125% dichromate solutions, respectively. Increasing dichromate concentration (0.5 to 2.5% Cr

solution concentrations) with 168 hr exposure did not result in increased Cr content in skin. Long lag times for appearance of Cr in the recipient phase combined with lack of increased skin concentration with time indicates a high binding capacity for Cr that will interfere with diffusion through the skin, although skin binding sites can eventually be exhausted with time. Gammelgaard et al. (1992) also observed the ratio of Cr(VI) to Cr(III) at pH 10 in the recipient phase to increase over 160 hr of exposure. Appearance of chromium as Cr(VI) in the recipient phase increased from about 60% at 40 hrs, to greater than 90% at 120 hrs. This finding indicated reduced capacity for dermal Cr(VI) reduction, eventually resulting in increased Cr(VI) passing through the skin.

Baranowska-Dutkiewicz (1981) found chromium (VI) from aqueous solutions to be readily absorbed by human skin. Seven volunteers were exposed to sodium chromate solutions (0.01, 0.1, and 0.2 M) on an area of the forearm for 15, 30 or 60 minutes, in a series of experiments. The exposure area was covered with a watch glass throughout the exposure period. Absorption was calculated from the difference between the applied and recovered dose of chromium (VI). The authors reported that percutaneous absorption of chromium is dependent on both concentration and time. Specifically, they found that (1) absorption was highest from the 0.01 molar solution (7.7-23% of applied dose) and lowest from the 0.2 molar solution (3.4-10.6% of applied dose), (2) the rate of absorption decreased as exposure time increased, and (3) the rate of absorption increased proportionally as exposure concentration increased. Individual data were not provided.

Wahlberg and Skog (1963) used disappearance measurements of radiolabeled chromium to estimate dermal absorption of hexavalent chromium in vivo in guinea pigs. Animals were exposed for 5 hours to various concentrations (0.00048 - 4.870 molar) of sodium chromate labeled with ^{51}Cr. Dermal absorption of chromium was confirmed qualitatively by organ analysis. The maximal disappearance of hexavalent chromium was observed from a 0.261 molar solution. Of the 10 animals exposed to this concentration, the mean disappearance percentage per 5-hour period was 4% of the applied dose.

No studies could be located that examined dermal uptake of Cr(VI) from soils. However, chromium fate in soil and soil bioaccessibility studies (gastrointestinal and sweat leaching) have been conducted.

The relationship between Cr(VI) and Cr(III) in soil is a dynamic one, which is affected by soil type and mineral content, pH, solubility, and other factors (Bartlett, 1991; Fendorf, 1995; Stewart et al., 2003). Cr(VI) exhibits greater mobility and less adsorption in soils compared to Cr(III). Organic matter, Fe(II), and sulfides in soils are capable of reducing Cr(VI) to Cr(III), while manganese oxides in soils are capable of oxidizing Cr(III) to Cr(VI). Usually, part of any Cr(VI) added to soil will be reduced instantly, especially under acid conditions. However, high concentrations of polluting Cr(VI) may quickly exhaust the readily

available reducing power of the matrix material and excess Cr(VI) may persist for years in soils without reduction.

Oral bioaccessibility of Cr(VI) from aged soils was determined by Stewart et al. (2003) using a physiologically based extraction test designed to simulate the digestive process of the stomach. It would be expected that bioaccessibility for dermal absorption of soil Cr(VI) would be no greater than oral absorption, and has been used to estimate dermal exposure to Cr(VI) in soil in previous health assessments (Sheehan et al., 1991).

In general, Cr(VI) bioaccessibility decreased with the aging of Cr(VI) in soils, with decreased bioaccessibility being most rapid for the first 50 days and then slowing dramatically between 50 and 200 days (Stewart et al., 2003). Chromium bioaccessibility was significantly influenced by reduction processes catalyzed by soil organic carbon. Soils with sufficient organic carbon had lower Cr(VI) bioaccessibility values of about 10 to 20% due to enhanced reduction of Cr(VI) to Cr(III). In soils where organic carbon was limited and reduction processes were minimal, considerably higher Cr(VI) bioaccessibility values of 60-70% were recorded.

Soil samples from two chromium waste sites that varied considerably in Cr(VI) concentration were extracted with a synthetic sweat solution to determine the potential for dermal bioaccessibility of Cr(VI) from contaminated soils (Wainman et al., 1994). The soils examined were contaminated with slag containing chromium from chromate and bichromate production facilities in New Jersey. One set of soil samples contained 710 µg Cr(VI)/g soil and contained chromate blooms, a thin layer of bright yellow crystals on the soil surface. Approximately 83% Cr(VI) was extracted in sweat from the soil with chromate blooms. Adjusting the pH of the soil from pH 5 to 8 had little effect on Cr(VI) extraction. In the other soil, the Cr(VI) concentration averaged 59 µg/g soil. Sweat extraction of Cr(VI) increased from 15 to 32% with increasing soil pH from pH 5 to 8. No Cr(VI) was extracted from the soil adjusted to pH 4. Extraction with distilled-deionized water was also performed, resulting in 76 and 27% extraction from soil with and without blooms, respectively.

Horowitz and Finley (1993) investigated the leaching of Cr(VI) in human sweat from chromite ore processing residue. The New Jersey ore residue originated from the same or similar processing facility as that investigated by Wainman et al. (1994). The human sweat at a pH of 7.2-8.0 extracted < 0.01% of Cr(VI) from the ore samples. Differences in the parent ore and extraction techniques were suspected to have led to the widely varying extraction of Cr(VI) from samples analyzed by Wainman et al. (1994) and Horowitz and Finley (1993).

Oral bioaccessibility studies have also been conducted on the New Jersey slag material (Hamel et al., 1999). Using two different methods, chromium in the slag

material had an average bioaccessibility of 34 or 40%, depending on the method used.

F. 3.4.2 Discussion and Recommendation for a Hexavalent Chromium (Soluble Compounds) ABS

In the comprehensive in vitro study by Gammelgaard et al. (1992), a measurable increase in Cr(VI) penetrating full thickness human skin could not be detected with 48 hr exposure and only 1.1% of Cr(VI) had been absorbed into the skin. By 190 hrs of exposure fractional absorption of Cr(VI) increased considerably to 15%. The in vitro data indicate Cr(VI) salts have a long lag phase and are slowly absorbed. In contrast, the in vivo human study by Corbett et al. (1997) suggests a very short lag time for appearance of Cr(VI) systemically, with increased Cr levels in the circulatory system within 3 hrs of immersion in a water tank of dilute aqueous dichromate. The human in vivo study by Baranowska-Dutkiewicz (1981) indirectly supports rapid dermal absorption of Cr(VI) with disappearance of aqueous Cr(VI) salt applied to skin for 15-60 min. Consequently, in vitro human exposure likely underestimates the dermal absorption potential of aqueous Cr(VI) solutions that occurs in vivo.

Alternatively, the indirect estimate of up to 23-44% dermal absorption of the applied dose of Cr(VI) salt by Baranowska-Dutkiewicz (1981) and Mali et al. (1964) likely overestimates the dermal absorption potential due to use of a skin occlusion application and reliance on a disappearance method to estimate absorption. Mali et al. (1964) found only 0.35 µg of chromium in stratum corneum tape stripping even though a total of 22 µg of Cr(VI) was assumed absorbed by disappearance from the skin surface. This finding does not correspond with data by Liden and Lundberg (1979) in which maximal levels of absorbed Cr(VI) was found in stratum corneum.

The 24 hr guinea pig in vivo study by Czernielewski et al. (1965) was the most comprehensive study available in regard to estimating whole body absorption of a dermally applied radiolabeled Cr(VI) solution. Analysis of excreta, blood, and most tissues yielded a fractional absorption of about 2.9%, of which 2.7% was found in excreta and blood. Dermal absorption in experimental animals often overestimates absorption in humans. The in vitro chromate disappearance constants for dermal exposures up to 24 hrs were 3-5 times greater through guinea pig skin compared to human skin (Wahlberg, 1965). However, recognizing that in vitro studies generate slower absorption rates of Cr(VI) than in vivo, the study by Czernielewski et al. (1965) provides a reasonable health protective absorption estimate (2.9%) when considering a human 48 hr in vitro fractional absorption of 1.1% was estimated by Gammelgaard et al. (1992).

To account for the effect of soil vehicle on dermal absorption of Cr(VI), the maximal Cr(VI) bioaccessibility of 83% in synthetic sweat as determined by Wainman et al. (1994) was taken into account. This bioaccessibility estimate

was from a soil sample with about 710 µg Cr(VI) per g soil and contained chromate crystals on the soil surface. The contaminated soil probably represents a matrix described by Bartlett (1991) in which high concentrations of Cr(VI) exhausted the readily available reducing power of the soil and excess Cr(VI) persists on the soil surface without being reduced. Thus, multiplying 2.9% by 0.83 and rounded to the nearest whole number provides an ABS point estimate of 2% for Cr(VI) from soil vehicle.

The Hot Spots risk assessment procedures have previously assumed no reduction of deposited Cr(VI) because typically Cr(VI) deposition is modeled without soil sampling monitoring for the Cr(VI)/Cr(III) ratio and without an evaluation of the redox potential of the soil. This assumption may result in overestimation of Cr(VI) soil concentrations in situations where Cr(VI) is readily reduced to Cr(III). Bioaccessibility is determined in part by the Cr(VI)/Cr(III) ratio. The use of soil with high concentrations of Cr(VI) to determine bioaccessibility is not likely to underestimate bioaccessibility under the conditions typically found in Hot Spots risk assessments, where Cr(VI) is deposited over a long period of time and typically results in lower soil concentrations than the 710 µg/g observed in the study by Wainman et al. (1994).

A Limitations for the ABS not discussed above include lack of a factor for absorbed chromium lost through skin desquamation. Studies show that some Cr(VI) will be reduced to Cr(III) in skin and bind to cellular constituents (Gammelgaard et al., 1992; Hostynek, 2003). If this occurs in the stratum corneum, the chromium will likely be removed through desquamation before systemic absorption can occur. Another limitation includes reliance on studies in which Cr(VI) is applied directly onto the skin (i.e., neat), rather than combined with soil, for estimation of fractional dermal absorption. Kissel (2011) has noted that fractional absorption is dependent on skin loading conditions for application of organic chemicals directly to skin. However, Baranowska-Dutkiewicz (1981) showed that for Cr(VI) the flux through skin increases proportionally with increasing Cr(VI) load applied to skin, resulting similar fractional absorption values independent of load onto skin. The constraints in estimating fractional absorption for organic chemicals applied neat, which assumes a constant flux through skin, does not appear to be relevant for the metal salt Cr(VI).

F.3.5 Fluoride and Soluble Fluoride Compounds

Recommended use of default inorganic compound ABS estimate of 13.0%.

F.3.5.1 Studies Considered

Excessive exposure to the negatively charged fluoride ion deposited on soil as an aerosol or as a soluble inorganic fluoride salt is known to have toxic effects in animals through ingestion of contaminated soil (Eagers, 1969). However, no quantitative data could be found regarding the fractional dermal absorption of

soil-bound fluoride or fluoride compounds following contact with skin. Two animal studies observed elevated fluoride serum levels or systemic toxicity following dermal exposure to concentrated hydrofluoric acid, but immediate skin corrosion was apparent and likely influenced dermal absorption (Derelanko et al., 1985; Boink et al., 1995).

Much of the fluoride naturally present in soils or deposited from facility emissions will generally be in, or strongly adsorbed to, soil particles and is not in a form accessible for uptake by the body (Davison, 1987). Highest levels of water-soluble, or bioaccessible, fluoride in heavily contaminated soils was about 15-20% of total fluoride (Polomski et al., 1982). Among several studies, the bioaccessible fluoride fraction in uncontaminated soils ranged from 0.06 to 7% of total soil fluoride (Gisiger, 1968; Polomski et al., 1982; Milhaud et al., 1989; Buykx et al., 2004).

F.3.5.2 Discussion and Recommendation for a Fluoride and Soluble Fluoride Compound ABS

Due to the lack of quantitative data regarding dermal absorption of soil-bound fluoride, it is not possible to determine an ABS from the data available. Use of a ~~1~~3% fractional absorption default value, based on the mean of the derived ABS values for the other Hot Spots metals and semi-metals (As, Cd, Cr(VI), Pb, Hg, Ni), will likely not underestimate dermal absorption of soil-bound fluoride~~,~~ given the highly ionic nature of fluoride and the strong adsorption of deposited fluoride to soil particles.

F. 3.6 Lead and Inorganic Lead Compounds

Recommended point estimate for dermal uptake: 3%

F. 3.6.1 Studies Considered

A. Key Study

The in vitro dermal absorption of lead oxide (PbO) powder (<10 µm particle diameter) in human abdominal skin was investigated (Filon et al., 2006). Each cell had a surface area of about 3.14 cm^2 and was filled with 5 mg PbO/cm^2 and with 2 ml synthetic sweat at pH 5.0. At 24 hrs, a median of 2.9 ng/cm^2 (0.06% fractional absorption) had penetrated the skin to the receiving solution and a median of 321.3 ng/cm^2 (6.4% fractional absorption) was absorbed in the skin following surface decontamination. In another experiment, removal of PbO after 30 min exposure did not cause a reduction of Pb penetration in 24 hrs, but did cause a reduction in skin Pb content. This finding suggested that initial rapid absorption of Pb can occur during the first few min of exposure.

B. Supporting Studies

Bress and Bidanset (1991) studied percutaneous absorption of lead in vitro using human abdominal skin obtained from autopsy, and guinea pig dorsal skin. PbO or lead acetate (10 mg) in saline solution was applied to 1.3 cm^2 skin samples. After 24 hours, the lead content of the saline reservoir fluid was measured. The lead content of the skin samples after exposure was not measured. In this experiment, 0.05% of the applied dose of lead acetate was recovered in the reservoir fluid, and less than 0.01% of the PbO. There was no difference between human and guinea pig skin.

Bress and Bidanset (1991) also examined in vivo percutaneous lead absorption in guinea pigs. Lead acetate or PbO, mixed in aqueous solution, was applied to a shaved area (2 cm^2) of the back (300 mg lead per kg body weight). After exposure for 1 week, the animals were killed and lead was measured in blood, brain, liver and kidney. Percent of applied dose absorbed could not be determined from this study. However, the concentration of lead in the measured tissues following lead oxide exposure was similar to that from control animals. In contrast, the lead concentration in measured tissues following lead acetate exposure was greater than controls, although absorption was considered poor, and statistics were not provided.

Moore et al. (1980) studied percutaneous absorption of lead acetate in humans from two commercial hair dye products. The products (one a lotion and one a cream) were spiked with lead-203 (^{203}Pb) and applied to each subject's forehead (n=8) for 12 hours. The preparations were applied in various forms (wet and dried) with periods of one month between each application. Lead absorption was estimated from blood counts, whole-body counts, and urine activity. Results were normalized for each subject by administration of an intravenous tracer dose of lead chloride.

The mean uptake of ^{203}Pb activity, measured in whole body at 12 hours, was greatest when the preparation was dried and skin was slightly abraded (0.18% of applied dose). The mean absorption including all methods of application (measured in whole body at 12 hours) was 0.058% with a range of 0-0.3%. It has been noted that the presence of colloidal sulphur in the lead acetate formulations used by Moore et al. (1980) may have led to the formation of insoluble lead sulfide, which would be unlikely to be significantly absorbed through skin (Stauber et al., 1994).

In a series of studies in human volunteers, aqueous solutions of inorganic lead salts including lead chloride and lead nitrate were shown to be rapidly absorbed through skin within 3-6 hrs and enter the extracellular compartment, resulting in increased concentrations of lead in the sweat and saliva but not the blood (Lilly et al., 1988; Stauber et al., 1994). However, application of radiolabeled lead (^{204}Pb) to skin of volunteers resulted in measurable increases of ^{204}Pb in the blood but with a very short residence time (Stauber et al., 1994). Preliminary experiments

also showed rapid absorption of lead oxide and elemental lead through the human skin of volunteers and detection in the sweat within a few hours. Only $PbCO_3$ was not absorbed through skin. In mice, skin-absorbed lead concentrated more strongly in skin and muscle, and less in blood and other organs compared to intravenously injected lead (Florence et al., 1998).

The authors proposed that the behavior of skin-absorbed lead in the body is different from lead that is ingested or injected, in that lead which passed through skin is in a physicochemical form with low affinity for erythrocytes and a high affinity for extracellular fluid compartments. The implication is that testing blood for lead exposure may not fully account for absorption of lead through the skin.

Stauber et al. (1994) examined dermal lead absorption by placing lead nitrate and lead nitrate spiked with ^{204}Pb on the arms of volunteers for 24 hrs. Rapid increases of lead were observed in sweat samples from the unexposed arm and in saliva, but only small concentrations of lead in blood and urine. However, high levels of ^{204}Pb in blood and urine were measured 2 and 16 days, respectively, after exposure ended suggesting slow absorption of lead into the blood from lead retained in the skin.

In order to quantify dermal lead absorption, 4.4 mg lead (as 0.5 M $Pb(NO_3)_2$) was dispensed onto filter paper and secured with plastic wrap to the left arm of one subject. After 24 hours, the filter paper was removed and the arm was washed. Of the 4.4 mg lead, 3.1 mg was recovered from the filter paper and wash fluid. Using this disappearance technique, the authors estimated that 29% of the lead was absorbed into or through the skin. In two volunteers, the estimated excretion of skin-absorbed ^{204}Pb in the sweat of two volunteers over 24 hrs was 16 and 46 µg lead/L. Assuming an average sweat production of 500 ml/day, the authors estimated 0.6% and 1.5% of the total lead that was absorbed was excreted in sweat.

Lead acetate or nitrate was also applied to the skin of mice by the researchers in order to quantitate the amount of lead absorbed and retained in organs and tissues (Florence et al., 1998). Forty µl of aqueous solutions of the lead salts (6.4 mg of lead) were applied to a shaved area of skin and covered with Parafilm. Mice were sacrificed and organs and tissues analyzed for lead content after time periods of 2 hrs to 1 week. A total analysis of the organs, feces, and urine showed that, of the 6.4 mg of lead applied to the skin, 26 µg (0.4%) was absorbed through the skin and entered the circulatory system in 21 hrs. This analysis does not appear to include skin-absorbed lead at the site of application. No differences in absorption of the two lead salts were observed. Increased organ content of lead was noted by 6 hrs of exposure, with maximal organ concentrations generally occurring after 24-48 hrs of exposure.

To investigate the stratum corneum depth profiles of lead in lead battery workers, 10 repeated skin strips were collected from exposed skin (dorsal hand) and

nonexposed skin (lower back) of 10 volunteers (Sun et al., 2002). Skin areas to be sampled were washed with soap and water, then ethanol, prior to collection in the morning before work. Total lead in stratum corneum strippings ranged from 20.74 to 86.53 µg (mean = 42.8 µg) from the hand, and 8.94 to 28.32 µg (mean = 17.4 µg) from the back. Approximately 20.8 µg (49%) of the total lead in the stratum corneum were in the first two tape strippings. There was a decreasing amount of lead content from both skin regions going from the outer to the inner layers, suggesting both regions had been contaminated with lead. Total amount of lead in the hand, but not the back, was linearly correlated with the amount of lead in blood. These findings indicate the source of lead in skin was from dermal exposure, rather than absorption of lead from the circulatory system into the skin.

Although the lead compound, which workers were exposed to, was not specified in the Sun et al. (2002) study, the primary lead compounds emitted during lead-acid battery production are identified as PbO and elemental lead (USEPA, 1998; Ruby et al., 1999). Elemental lead particles that are deposited in soils quickly form coatings of highly bioavailable PbO.

The leaching behavior of lead-contaminated soil can be divided into three stages based on the leachate pH: a high alkalinity leaching stage at pH > 12, where Pb formed soluble hydroxide anion complexes and leached out; a neutral to alkaline immobilization stage in the pH range of 6-12, which was characterized by low Pb leachability by adsorption and precipitation; and an acid leaching stage with pH < 6, where leachability increased exponentially with decreasing pH and was characterized as free Pb-ion (Jing et al., 2004). This study indicates that soluble Pb at the neutral pH found in most soils would only be a fraction of the total Pb content of the soil.

Several leaching studies of Pb-contaminated soils suggest the bioaccessible Pb in soil can vary greatly. Within a pH range of 7-8, soluble Pb ranged from less than 0.01% to 48% of total Pb content of soil (LaPerche et al., 1996; Yang et al., 2001; 2002; Jing et al., 2004). In a major Pb contamination due to a paint spill the Pb soil content was 34,592 mg/kg, which is roughly an order of magnitude greater than many Pb-contaminated soils (Zhang et al., 1998). Soluble Pb at pH 7 was roughly estimated to be 18% of total soil Pb. At pH 5, fractional soluble Pb increased to about 41% of total soil Pb.

F. 3.6.2 Discussion and Recommendation for a Lead and Inorganic Lead Compound ABS

The accumulated in vivo absorption data did not provide enough quantitative information to estimate an ABS point estimate of lead including both systemic absorption and that retained in skin. Additionally, no data could be found that measured dermal absorption of lead from contaminated soil. Thus, the lead ABS point estimate incorporated data from an in vitro human study of lead applied neat and soil leaching tests for lead-contaminated soil.

The most comprehensive human data available were the in vitro study by Filon et al. (2006), which observed 0.06% of applied lead penetrating to the receiving solution and 6.4% of applied lead retained in skin following dermal exposure of PbO in a synthetic sweat solution. The skin depth profile of lead shows 49% of the total lead in the stratum corneum was in the first two tape strippings, and might be removed through desquamation prior to systemic absorption (Sun et al., 2002). However, human in vivo dermal exposure data suggest a relatively short lag time for appearance of lead in blood and continual absorption of lead into the blood from the skin reservoir (Lilly et al., 1988; Stauber et al., 1994). Until further studies are conducted to estimate the fraction of lead removed via desquamation prior to systemic absorption, it is presumed that all the lead absorbed in skin is available for systemic absorption.

Although only 0.06% of the lead reached the receiving solution in the in vitro study by Filon et al. (2006), in vitro dermal absorption studies of metal salts generally do not include a full accounting of absorption due to skin shunts such as hair follicles and sweat ducts. Hostynek (2003) noted that these skin shunts swell shut upon hydration during in vitro dermal absorption studies, and can reduce the movement of some dermally applied metal salts directly into lower skin layers. The human in vivo data support the importance of sweat ducts for lead dermal absorption (Lilly et al., 1988; Stauber et al., 1994). In addition, the rapid reduction of lead dermal absorption early during exposure in the Filon et al. (2006) in vitro study has been considered evidence for skin shunts becoming hydrated and reducing lead absorption by these pathways (Hostynek, 2003). These data further support the reasoning that the lead retained in skin observed by Filon et al. (2006) cannot be discounted for potential systemic absorption.

In soil, aqueous leaching studies suggest soluble Pb can vary greatly depending on the soil characteristics. If sweat is the leachate, the pH can range between 4 and 7, with an average in male Caucasians of 4.85 (Wainman et al., 1994). The acidic nature of sweat will likely enhance Pb bioaccessibility from soil compared to the soil pH ranges of 7-8. Because of the wide range of solubilities of Pb in soil, a health protective point estimate based on the solubility of a heavily Pb contaminated soil at pH 5 (average pH of sweat) is warranted. Zhang et al. (1998) observed an approximate 41% Pb solubility at pH 5 from soil that may have been saturated with Pb paint (Pb content = 34,592 mg/kg soil). Adjusting the total fractional dermal absorption of 6.46% observed by Filon et al. (2006) by multiplying the fraction of soluble Pb in a highly impacted soil (0.41) determined by Zhang et al. (1998) results in an ABS point estimate of 3% after rounding to the nearest whole number.

~~A health protective~~The ABS of 3% for Pb salts is higher than ~~the~~ most other metal salts investigated. However, most of the soil leaching experiments used soils that were environmentally contaminated or incorporated time as a factor to control for soil aging. Absorption of Pb salts has also been shown to be high by

the oral route relative to other metals, up to 90% absorption in the acidic environment of the stomach (Ruby et al., 1999). A limitation for this ABS is the reliance on studies in which lead is applied neat to skin, rather than combined with soil, for estimation of fractional dermal absorption. Kissel (2011) has noted that fractional absorption is dependent on skin loading conditions for application of organic chemicals directly to skin. However, Baranowska-Dutkiewicz (1981) showed that for Cr(VI) the flux through skin increases proportionally with increasing Cr(VI) load applied to skin, resulting similar fractional absorption values independent of load onto skin. Thus, metal salts of lead applied neat probably adhere closer to the dermal absorption kinetics of Cr(VI), rather than to organic compounds.

F. 3.7 Inorganic Mercury Compounds

Recommended point estimate for dermal uptake from soil: 3%

F. 3.7.1 Studies Considered

Quantitative in vivo dermal absorption studies of Hg-contaminated soils have not been performed. A summary of the in vitro dermal studies exposing human and animal skin to Hg-contaminated soil are shown in Table F-2.

A. Key Studies

The dermal bioavailability of $^{203}HgCl_2$ was tested in vitro on dermatomed male pig skin as pure compound or following addition to sandy soil or clay soil (Skowronski et al., 2000). The Yorkshire pig model was chosen due to histological, physiological, biochemical and pharmacological similarities to human skin. The sandy and clay soil consisted of 4.4% and 1.6% organic matter, respectively, and a majority of the soil particles were in the range of 50-250 µm. A soil loading of 47 mg/cm^2 was calculated from the data provided and the $HgCl_2$ concentration was 5.3 ng/mg soil. Absorption was estimated up to 16 hrs following application.

In general, dermal absorption of Hg was greater from sandy soil than from clay soil. In both soils, the rate of appearance of Hg in the receptor fluid was rapid during the first hour, then decreased to a steady state for the remaining 15 hrs. In sandy soil freshly spiked with Hg, 0.28% and 37.5% of the applied dose had penetrated the skin to the receptor fluid and was bound to skin, respectively, at 16 hrs. In clay soil freshly spiked with Hg, 0.08% and 39.7% of the applied dose had penetrated the skin to the receptor fluid and was bound to skin, respectively, at 16 hrs. For the pure compound, Skowronski et al. (2000) observed a skin penetration of 0.18%, but the amount bound to skin was 66.3%. For Hg aged 3 months in soil, dermal absorption was reduced to 3.3% in sandy soil and 2.6% in clay soil. Only 0.04% and 0.01% of these totals in the sandy and clay soil,

respectively, represented percent of applied dose penetrating to the receptor fluid.

B. Supporting Studies

Radiolabeled mercuric chloride ($^{203}HgCl_2$) was mixed with soil and applied in vitro onto fresh human breast skin (obtained within 24 hrs of harvest) for 24 hrs by means of Bronaugh diffusion cells (Moody et al., 2009b). The same amount of $^{203}HgCl_2$ was also applied without soil to human skin samples. The soil had been sieved to 90-710 µm prior to spiking with the Hg salt. The soil mixture (3.2 mg soil) was added to the diffusion cells resulting in a soil loading of 5 mg/cm^2. At 24 hrs, mean percent dermal absorption including the skin depot was 46.6 and 78.3% with and without soil, respectively. The fraction of total absorbed Hg that entered the diffusion cell in 24 hrs was 1.5 and 1.4% with and without soil, respectively.

A radiolabeled mercury compound ($^{203}HgCl_2$) was applied in soil or water vehicle to human skin in vitro (0.5 µg/cm^2 containing 1 µCi) for 24 hours (Wester et al., 1995; Wester and Maibach, 1998c). The investigators used Yolo County soil (26% sand, 26% clay, 48% silt, 0.9% organic) sieved for 180-300 µm particles. Receptor fluid accumulation from either water vehicle or soil vehicle was 0.07% of applied dose. Previously frozen or fresh skin gave similar results. Skin content of mercury from water vehicle averaged 29% of total dose applied. Using soil loads of 5, 10, and 40 mg, skin content of mercury was 10.4, 6.1, and 7.2% of dose applied, respectively.

In other human in vitro studies by the same research group, 5.5% absorption into skin and 0.01% penetration of pure $HgCl_2$ into receptor fluid was observed with a 30 min exposure (Wester et al., 1995; Wester and Maibach, 1998c). Continued perfusion for 48 hrs following the 30 min exposure increased skin absorption and penetration to receptor fluid to 6.3% and 0.09%, respectively, exhibiting the ability of Hg to migrate through skin after removal of Hg from the skin surface. When the in vitro exposure was increased from 30 min to 24 hrs, mercury skin absorption and penetration to receptor fluid was increased to 35.4% and 0.06%, respectively. No other results or methodology details were provided.

The dermal bioavailability of liquid and soil-bound $^{203}HgCl_2$ was tested on dermatomed human male skin in vitro (Sartorelli et al., 2003). For the liquid vehicle, $HgCl_2$ was added to buffered water solution (pH = 4.0). For the soil vehicle, $HgCl_2$ was added to loam soil consisting of 60% sand, 30% silt and 10% clay sieved to a particle size of <150 µm. Soil loading on skin was about 40 mg/cm^2, which would be greater than monolayer coverage using a particle size of <150 µm. The concentration of $HgCl_2$ was 0.0069 or 0.1190 nmol/cm^3. After 72 hr exposure, any mercury absorbed from soil and penetrating skin to the receiving fluid was below the detection limit. Mean mercury concentrations in the skin were 10.53% of the applied low dose and 15.04% of the applied high dose.

Mercury in the liquid vehicle was also applied at two concentrations of 0.0088 and 0.0607 nmol/cm^3. At the low dose, percent of applied dose penetrating skin to the receptor fluid was 1.64 and 4.80% at 24 and 72 hrs, respectively. At the high dose, percent of applied dose penetrating skin to the receptor fluid was 0.34 and 0.93% at 24 and 72 hrs, respectively. Percent of applied dose retained in skin at 72 hrs was 18.93 and 44.97% for the low and high dose, respectively.

TABLE F.2. In Vitro Dermal Absorption Results of Mercuric Chloride from Soil

Study	Species	Exposure time (hr)	Soil fraction (µm)	% Reaching receptor	% Total absorbed fresh	% Total absorbed aged
Skowronski et al., 2000	pig	16	unsieved	0.28 [a] 0.08 [b]	37.8[a] 39.8[b]	3.3[a] 2.5 [b]
Moody et al., 2009	human	24	90-710	1.5	46.6	ND[c]
Wester et al., 1995	human	24	180-300	0.07	7.9	ND
Sartorelli et al., 2003	human	72	<150	0[d]	13	ND

[a] Sandy soil
[b] Clay soil
[c] Not determined
[d] Below the limit of detection

Hursh et al. (1989) studied dermal absorption of mercury vapor in humans. Each of 5 men exposed the skin of one forearm (a single exposure) to vapors with concentrations ranging from 0.88-2.14 ng $^{203}Hg/cm^3$ for periods of 27 to 43 minutes. The rate of dermal uptake of mercury by the arm was quantified by measuring the difference between accumulated radioactivity on exposed and unexposed forearms following exposure. The mean uptake rate for the 5 subjects was reported as 0.024 ng Hg per cm^2 skin per minute per ng Hg per cm^3 air. At this rate, the authors estimate that dermal absorption of mercury from vapor is approximately 2.6% of the rate of uptake by the lung.

In addition, the study protocol by Hursh et al. (1989) included a procedure in which adhesive strips were applied every 3-4 days post exposure for up to 40 days, which regularly removed cells of the stratum corneum from the same marked skin area following exposure. Larger amounts of Hg were stripped at later time points, suggesting that a substantial fraction of the absorbed Hg was probably associated or bound to keratinocytes rather than stratum corneum. Based on the whole body count of radiolabeled Hg and the amount of Hg absorbed in the skin, the authors note that about half of the Hg eventually reached the bloodstream while the remainder was shed by desquamating cells. The data show estimates of 26, 43, 45, 45 and 46% of the dermally absorbed Hg reaching the bloodstream in the five volunteers. It was theorized that the elemental Hg penetrated the stratum corneum as vapor but that in the epidermis,

some, but not all, of the Hg became oxidized to mercuric ions. The ions then became fixed or bound in the skin, some of which then moved upward and was eventually shed.

Baranowska-Dutkiewicz (1982) exposed the forearms of eight male volunteers to aqueous mercuric chloride solutions. Aliquots (0.25 ml) of $HgCl_2$ solutions were applied directly to a 22 cm^2 area of skin and covered with a watch-glass. Percutaneous absorption of mercury was calculated as the difference between the amount applied and the amount recovered after the skin and the watch-glass were washed. In order to examine the effect of concentration on uptake, 3 concentrations (0.01, 0.1, and 0.2 M) were applied for 30 minutes. As concentration increased, rate of uptake increased. In order to examine the influence of exposure time on uptake, 0.1 M $HgCl_2$ was applied for 5, 10, 15, 30 and 60 minutes. The authors reported that the average rate of uptake of mercury decreased from 9.3 µg/cm^2/min during a 5 minute exposure, to 2.5 µg/cm^2/min during a 1 hour exposure. The average percutaneous absorption of mercury was calculated for exposures of 5, 10, 15, 30, and 60 minutes resulting in 20%, 29%, 37%, 60% and 64% absorption of the applied dose, respectively.

In vivo application of aqueous $HgCl_2$ (0.1% w/v) to normal human skin followed by biopsy and visualization with electron microscopy found mercury deposits present intracellulary and extracellularly in the stratum corneum within minutes after application (Silberberg, 1972). The presence of mercury in the epidermis was not apparent until 2-4 hrs after application. The finding of immediate absorption of $HgCl_2$ correlates well with the in vivo findings of Baranowska-Dutkiewicz (1982), which observed the disappearance of $HgCl_2$ within 5 min after application to human skin.

An in vivo study in guinea pigs found that dermal absorption of Hg from $HgCl_2$ steadily decreases with increasing dose, suggesting a build up of a secondary diffusion barrier as a consequence of the electrophilic metal forming irreversible bonds with proteins of the skin (Friberg et al., 1961). Thereby a depot accumulates in the stratum corneum retarding further penetration in inverse proportion to metal concentration. This secondary barrier build-up retarding absorption was also evident with increasing dermal exposure intervals. $HgCl_2$ applied in vitro on human skin showed greatest percutaneous absorption during the first 5 hrs (Wahlberg, 1965). With later time periods the absorption rate decreased. The average absorption rate over the first 24 hrs was only about one-fourth the rate observed during the first 5 hrs of dermal exposure.

F. 3.7.2 Discussion and Recommendation for an Inorganic Mercury Compound ABS

More than 98% of mercury in soils is present as nonalkyl Hg(II) compounds and complexes, with direct deposition a significant component for much of the loading to terrestrial soils (Davis et al., 1997). In the soil, Hg can occur in three different

valence states, namely as Hg^0, Hg_2^{2+} and Hg^{2+} (Andersson, 1979). Hg^{2+} forms various complexes with OH^- and Cl^- ions, with the dominating mercuric complexes being $HgCl_2$, $Hg(OH)_2$ and HgOHCl. Only a small fraction of mercuric Hg species occurs free in solution; the major fraction is either bound to or in the soil material. Hg^{2+} and gaseous Hg^0 forms are preferably bound to organic matter in acidic soils, whereas in neutral and slightly alkaline soils, mineral components are active as well. Mercury exhibits a very high affinity for sulfide in reducing environments, forming relatively insoluble HgS (Davis et al., 1997).

Human skin both in vivo and in vitro has been shown to have a large capacity to accumulate metallic mercury vapor or mercury salts (as $HgCl_2$) applied in aqueous solution directly to skin. When freshly mixed with soil, Hg salts appear to have a greater ability for absorption into skin than other metal salts of concern in this section (i.e., Ni, Pb, Cd, etc.). However, similar to other metals, aging of Hg salt in soil significantly reduces the fractional absorption of Hg into skin. Therefore, a fractional absorption of 3% for $HgCl_2$ aged in soil prior to testing was chosen as the basis of the ABS to account for the aging affects in soil.

The Hg ABS is based on the in vitro study in pigs by Skowronski et al. (2000), in which $HgCl_2$ aged in soil for three months resulted in a considerable reduction of fractional absorption compared to $HgCl_2$ freshly mixed with soil. Limitations of this study include use of skin from a non-primate species, less than 24-hr exposure, and likely exceedance of soil monolayer coverage during the exposure. However, the human in vitro studies shown in Table F-2 also have their limitations for estimating fractional absorption, including exceedance of soil monolayer coverage (Sartorelli et al., 2003), or use of soil fractions that do not include soil particles less than 90 to 180 µm, which most commonly adhere to skin (Wester et al., 1995; Moody et al., 2009b).

Given the limitations, it is still unlikely that the ABS will underestimate fractional absorption. While both the human and animal in vitro studies show a large capacity for dermal absorption of Hg salt, very little reaches the diffusion cells (see Table F-2). Other studies reviewed here indicate that some of the Hg^{++} ions in mercuric salts tend to bind tightly to cellular proteins in all strata of skin, including stratum corneum, which may then impede further diffusion of mercury (Friberg et al., 1961; Silberberg, 1972; Hostynek, 2003). Mercury bound in stratum corneum would likely be removed via desquamation of skin. Hursh et al. (1989) have shown that a considerable portion of absorbed Hg in skin will eventually be lost (up to 50%) due to desquamation.

Nevertheless, the development of a Hg ABS would benefit from human in vitro studies with Hg salts aged in soil, and continued monitoring after 24-hr dermal exposure to better estimate the amount of Hg that reaches the circulation (i.e., reaches the diffusion cells) and how much is likely to be lost due to desquamation. Because the ABS is based on Hg aged in soil, the ABS may

underestimate fractional dermal absorption for soils in which a significant fraction of Hg has been very recently deposited on soil, or for soils that are heavily contaminated or saturated with Hg.

F. 3.8 Nickel and Nickel Compounds

Recommended point estimate for dermal uptake from soil: 4%

F. 3.8.1 Studies Considered

A. Key Studies

Radiolabeled nickel chloride ($^{63}NiCl_2$) was mixed with soil and applied in vitro onto fresh human breast skin (obtained within 24 hrs of harvest) for 24 hrs by means of Bronaugh diffusion cells (Moody et al., 2009b). The same amount of $^{63}NiCl_2$ was also applied without soil to human skin samples. The soil had been sieved to 90-710 µm prior to spiking with nickel salt. The soil mixture (3.2 mg soil) was added to the diffusion cells resulting in a soil loading of 5 mg/cm^2. At 24 hrs, mean percent dermal absorption including the skin depot was 1 and 22.8% with and without soil, respectively. The fraction of total absorbed nickel that entered the diffusion cell in 24 hrs was 0.5 and 1.8% with and without soil, respectively.

In vivo, sequential adhesive tape stripping was implemented to characterize the penetration of nickel salt solutions in methanol and nickel metal powder in human stratum corneum following 24 hr occlusive application to the forearm (Hostynek et al., 2001a; Hostynek et al., 2001b). Hostynek et al. (2001a) investigated stratum corneum depth profiles for chloride, sulfate, nitrate and acetate nickel salts. Penetration of the stratum corneum by nickel salts at levels of 0.001-1% nickel salt was limited and closely related to the counter ion. The total percent dose of each salt recovered in stratum corneum was 26.1, 18.5, 8.8, and 3.3% for the nitrate, acetate, sulfate, and chloride, respectively. Tape stripping of the skin showed that most of the dose remained on the surface or was retained in the superficial layers of the stratum corneum. Depth profiles converged towards non-detectable levels in the lower stratum corneum regardless of concentration for the acetate, chloride and sulfate. Nickel applied as nitrate is retained at a constant level of approximately 1% of applied dose in the lower layers of the stratum corneum.

The in vitro permeation of 1% aqueous solutions of chloride, sulfate, nitrate, and acetate nickel salts across only the stratum corneum was investigated using human leg skin (Tanojo et al., 2001). An initial surge in permeation rate within the first 24 hrs was observed for the nickel salts, followed by steady-state permeability rate up to 96 hrs that was not significantly different among the four salts. Nickel sulfate penetration of stratum corneum was greatest at 1.09%, whereas nickel nitrate recovery within stratum corneum was greatest at 0.95%.

Total absorption (receptor fluid plus bound to stratum corneum) was 1.65, 1.49, 0.92, and 0.12 % for the sulfate, nitrate, chloride, and acetate salts, respectively. Total recovery of absorbed and unabsorbed nickel was virtually complete for all the salts except nickel nitrate, in which 84% recovery was attained.

Permeation of the salts was attributed by Tanojo et al. (2001) solely to the diffusion across the transcellular/intercellular barrier, as hair follicle and gland shunts were shut upon hydration by the aqueous solutions. These pathways swelling shut early during in vitro exposure may explain the decreased rate of absorption of nickel following an initial surge. Lack of ability to account for absorption of nickel via skin shunts may underestimate absorption.

B. Supporting Studies

Nickel reversibly binds to constituents of the epidermis when human epidermis was homogenized and incubated with nickel chloride solutions (Fullerton and Hoelgaard, 1988). Spruit et al. (1965) utilizing human cadaver skin has shown that nickel ions also reversibly bind to the dermis. Nickel powder has also been shown to oxidize when suspended in synthetic sweat, whereupon the metallic ions can be absorbed in vitro through human skin (Larese et al., 2007).

Under the same experimental exposure conditions as used by Hostynek et al., (2001a), nickel metal powder (particle size 3 µm) values were found to decrease from the superficial to the deeper layers of the stratum corneum (Hostynek et al., 2001b). However, nickel was still present at the deepest levels of stratum corneum removed by adhesive stripping, indicating that the metal has likely reached the viable epidermis and has potentially become systemically available. Although the data did not lend itself to estimation of a skin permeation rate, total nickel removed with 20 strips from the skin after 24 hr occlusion with 21.7 mg/cm^2 nickel powder was 38.7 µg/cm^2 (i.e., approximately 0.18% of the total nickel metal applied was found in the stratum corneum). These data indicated that in intact skin, nickel metal is oxidized to form soluble, stratum corneum-diffusible compounds which penetrate the intact stratum corneum.

Dermal absorption of nickel chloride as $^{63}NiCl_2$ from two different soils was determined in vitro through dermatomed pig skin cut 200 µm thick (Abdel-Rahman et al., 1997). Soil types included a sandy soil with 4.4% organic matter and a clay soil with 1.6% organic matter. Skin applications included $^{63}NiCl_2$ added immediately after the addition of the two soils (30 mg each) to skin, or after each soil was aged for 6 months with $^{63}NiCl_2$. Nickel chloride was also added alone in ethanol vehicle to separate skin samples. The chemical dose was 113.8 ng/cm^2 and the soil loading was calculated to be 47 mg/cm^2. Monolayer coverage was probably exceeded with a soil loading of 47 mg/cm^2, causing a reduction in the observed fractional absorption.

Following 16 hrs of exposure, 0.3% of freshly applied $^{63}NiCl_2$ in clay soil penetrated the skin to receptor fluid and 12.1% was found bound to skin. No significant difference for dermal absorption from sandy soil was observed. For the nickel solution applied to skin, 0.4 and 57.9% of the dose applied was found in receptor fluid and bound to skin, respectively. In aged sandy and clay soil, 0.03 and 0.05% nickel was found in the receptor fluid, respectively. Only 3.1 and 3.7% of the metal was bound to skin from sandy and clay soil, respectively. Aging nickel in the soils appeared to be complete by 3 months, as further aging in soil for 6 and 12 months did not result in further decreased dermal bioavailability of the metal (Abdel-Rahman et al., 1997; Abdel-Rahman et al., 1999).

Fullerton et al. (1986) examined the permeation of nickel salts, specifically nickel sulfate and nickel chloride, through human full-thickness breast or leg skin in vitro. Skin excised in surgery was exposed to aqueous solutions of 184 $\mu g/cm^2$ for each nickel salt for up to 144 hrs. In the first experiment the effect of occlusion on the permeation rate of nickel chloride was examined. Occlusion resulted in a significantly higher permeation rate (approximately 3.6 percent of applied dose) compared with non-occluded exposure (approximately 0.23 percent) after 144 hours.

In the second experiment, nickel ions from a chloride solution were found to pass through the skin about 50 times faster than nickel ions from a sulfate solution. The amount of permeation of nickel chloride was much higher (16%) at 144 hours than nickel sulfate (0.3%). However, dermal penetration of the skin was slow, having a lag-time of about 50 hours. The occluded-skin permeation of nickel chloride was considerably higher in experiment 2 than experiment 1 (9-16% vs 3.6%) and was attributed by the authors to the use of breast skin from different donors.

In another study by the researchers, the stripping method was used in vitro on human full thickness skin following exposure to 5% nickel chloride in a 5% methyl cellulose gel for 96 hrs under occlusion (Fullerton et al., 1988). Nickel penetration from the gel solution gave similar results to nickel penetration of the pure nickel salt. Skin depth profiles found 50.9% was present on and in the stratum corneum (skin was not washed before stripping) with most of the nickel in the upper part of the stratum coeneum, 10.6% in the epidermis, 1.6% in the dermis, and only 0.4% reached the receptor solution.

Although the time frame and doses were different, similar dermal absorption results were obtained by Turkall et al. (2003) with in vitro dermal exposure of pig skin to 64 ng of radiolabeled nickel chloride. Penetration of ^{63}Ni in ethanol through pig skin was 0.4% of initial dose and a total of 58% of the nickel remained in the skin at the end of 16 hrs.

F. 3.8.2 Discussion and Recommendation for a Nickel and Nickel Compound ABS

The only study that exposed human skin to soil contaminated with a nickel salt was the in vitro study by Moody et al. (Moody et al., 2009b). However, there is evidence to suggest in vitro tests for dermal absorption of nickel may underestimate absorption in vivo.

Hostynek et al. (2001a) observed a range of 26.1% to 3.3% absorption of applied dose over 24 hrs among four nickel salts tested in vivo on human stratum corneum. However, Tanojo et al. (2001) observed only a range of 1.65% to 0.12% absorption of applied dose over 96 hrs among the same four nickel salts tested in vitro on human stratum corneum. Comparison of these data indicates that reliance on in vitro absorption data probably underestimates the in vivo dermal absorption of nickel salts.

Specifically regarding the nickel chloride salt applied directly to skin, Hostynek et al. (2001a) observed a 24-hr total absorption of 3.3% for human skin in vivo, while Tanojo et al. (2001) observed a 96-hr total absorption of 0.92% for human skin in vitro. These data together suggests a 3.6-fold greater absorption in vivo compared to in vitro absorption.

Although the dermal absorption time used by Tanojo et al. (2001) was 96 hrs, most of the $NiCl_2$ had penetrated the skin in the first 24 hrs (probably greater than 95%) and appearance of nickel into the diffusion cells had attained steady state. Assuming steady state levels of $NiCl_2$ had also been reached in stratum corneum by 24 hrs, it can be estimated that the total absorption of $NiCl_2$ recorded by Tanojo et al. at 96 hrs was similar to that found at 24 hrs.

Applying a 3.6-fold in vivo/in vitro ratio adjustment to the fractional dermal absorption value of 1% for $NiCl_2$ determined by Moody et al. (2009b) results in an ABS value of 3.6% (or 4% when rounded to the nearest whole number). The ABS is similar to the fractional dermal absorption of 2-4% resulting from exposure of pig skin to $NiCl_2$ aged in different soils (Abdel-Rahman et al., 1997; Abdel-Rahman et al., 1999).

F. 3.9 Selenium and Selenium Compounds

Recommended use of default inorganic compound ABS estimate of ~~1~~3.0%.

F. 3.9.1 Studies Considered

No quantitative data could be found regarding the fractional dermal absorption of soil-bound selenium (Se) or Se compounds applied to skin.

In dermal absorption studies of Se solutions, Farley et al. (1986) applied a 2.5% selenium sulfide lotion topically overnight on human volunteers. Skin region exposed and surface area covered were not described. Se levels in urine following exposure were significantly increased over control levels, but absorption was considered too slight to result in toxic effects. Repeated overnight treatments in a few volunteers over two days did not result in Se concentrations in the urine which were significantly higher than normal. In another study, increased serum levels of Se could not be measured in human volunteers that applied 2.5% selenium sulfide lotion to their torso overnight (Kalivas, 1993). Used in shampoo as a 1% selenium sulfide concentration, weekly use for a year did not change the normal urinary Se level (Cummins and Kimura, 1971).

Selenium sulfide is insoluble in water and is considerably less toxic via the oral route compared to elemental selenium or ionic forms of water-soluble selenite and selenate salts, such as sodium selenite (Cummins and Kimura, 1971). Lower gastrointestinal absorption of the sulfide salt was thought to be the cause of the lower oral toxicity.

The fraction of applied dose of ^{75}Se internally absorbed following application of selenous acid, a highly water soluble Se compound, onto the pelts of rats was calculated to be 1% per day over a 9-day exposure period (Medinsky et al., 1981).

F. 3.9.2 Discussion and Recommendation for a Selenium and Selenium Compounds ABS

Due to the lack of quantitative data regarding dermal absorption of soil-bound Se compounds, it is not possible to determine a chemical-specific point estimate ABS. However, use of a ~~1~~3% fractional absorption default value for Se and Se salts for screening purposes, based on the mean of the derived ABS values for the Hot Spots metals and semi-metals (As, Cd, Cr(VI), Pb, Hg, Ni), will likely not underestimate dermal absorption of soil-bound Se, given that fractional absorption of highly soluble selenous acid applied neat to the pelts of rats was about 1% of applied dose.

F.4 Point Estimates for Dermal Absorption (ABS) of Organic Compounds

F. 4.1 Polychlorinated Biphenyls (PCBs)

Recommended point estimate for dermal uptake from soil: 14%

F. 4.1.1 Studies Considered

A. Key Study

The dermal uptake of each of the two commercial PCB formulations Aroclor 1242 and Aroclor 1254 was studied in vivo in female rhesus monkeys (Wester et al., 1993b). Aroclor 1242 is dominated by the tri- and tetra congeners (68 percent) and Aroclor 1254 is dominated by the penta- and hexa congeners (83 percent). Each PCB preparation was adsorbed onto soil particles that before sieving contained 26% sand, 26% clay, 48% silt, and 0.9% organic carbon. The soil was fractionated by particle size to 180 - 300 μm. The soil levels of the PCB preparations were 44 ppm Aroclor 1242 and 23 ppm Aroclor 1254.

The PCB laden soil was applied for 24 hours to a 12 cm^2 area of lightly shaved abdominal skin which was protected by a non-occluded patch. The applied doses were 1.75 μg/cm^2 Aroclor 1242 and 0.91 μg/cm^2 Aroclor 1254. The soil loadings were 40 mg soil/cm^2 skin for both preparations. Following the first 24 hour exposure during which systemic absorption was measured as the content recovered in urine and feces, the patch was removed, the visible soil was removed from the site of application, the treated skin was washed with soap/water, and urine/feces were collected for an additional 34 days. One group of monkeys was exposed to the PCBs intravenously to adjust the cumulative urine/feces recovery of the dermally applied PCBs. The corrected fractional dermal absorption was 13.9% for Aroclor 1242 and 14.1% for Aroclor 1254.

B. Supporting Studies

PCBs are frequently found as complex mixtures of isomers in soil. To determine the effect of chlorine substitution on dermal absorption, Garner and Matthews (1998) applied dermal doses of ^{14}C-labeled mono-, di-, tetra-, and hexachlorobiphenyls to 1 cm^2 areas on the backs of rats for 48 hrs. Dermal penetration varied inversely with the degree of chlorination and ranged from essentially 100% for monochlorobiphenyl to about 30% for the hexachlorobiphenyl. However, the highly chlorinated PCBs tend to have slower metabolism and elimination and remain in the site of exposure longer, resulting in slow diffusion to the systemic circulation.

Mayes et al. (2002) dermally exposed female rhesus monkeys to radiolabeled Aroclor 1260 in soil in a manner similar to that used by Wester et al. (1993b). The soil was classified as sandy silt made up of 20% sand, 54% silt and 20% clay with a total organic carbon content of 5-6%. Sieving to <150 μm prior to application adjusted the total organic carbon content up to 8.7%. Five-hundred mg of soil either freshly spiked or aged for 88 days with PCBs (about 70 μg PCBs/g soil) was applied to a 12 cm^2 area of the chest/abdominal area and protected by a non-occluded patch. The calculated dermal load was 42 mg/cm^2. One group was exposed to radiolabeled PCBs intravenously to adjust the cumulative urine/feces recovery of dermally applied PCBs. Groups exposed for 12 or 24 hrs to PCBs aged in soil exhibited percutaneous absorption values of

3.43 and 4.26%, respectively, while a group exposed for 24 hrs to soil freshly spiked with PCBs exhibited a dermal absorption value of 4.07%.

Mayes et al. (2002) stated that the reduction in fractional absorption compared to the Wester et al. (1993b) study was due to greater soil content of organic matter, which absorbs highly lipophilic compounds such as PCBs. However, the dermal load of 42 mg/cm^2 used by Mayes et al. likely exceeded monolayer coverage and caused a reduction in fractional absorption. No statistically significant difference was observed between the 12- and 24-hr exposure groups, suggesting PCBs partition quickly into lipid components of the stratum corneum. Likewise, aging of PCBs in soil had no effect on dermal absorption, suggesting rapid binding to the organic fraction of soil. The authors noted that Aroclor 1260 has a slightly higher octanol/water partition coefficient (log K_{ow}) than Aroclors 1242 and 1254 used by Wester et al. (1993b). A higher log K_{ow} would favor greater dermal absorption. However, the higher percentage of congeners with seven or more chlorines in Aroclor 1260 compared to Aroclors 1242 and 1254 tends to reduce dermal absorption, as shown by Garner and Matthews (1998).

The dermal absorption of radiolabeled 3,3',4,4'-tetrachlorobiphenyl (TCB) from liquid and soil mixtures was studied in an ex-vivo Yorkshire-Landrace pig-skin-flap model (Qiao and Riviere, 2000). The soil was described as a dust containing 31.2% sand, 16.8% silt, 53.0% clay (90% kaolinite) and 0.3% organic matter. No particle size fractionation was given. Sixty-five to 70 mg soil containing 200 µg of ^{14}C-TCB (40 µg/cm^2) was applied onto 5 cm^2 skin surface for 8 hrs, and the area was either left open (non-occlusive) or closed with Parafilm (occlusive). Greatest dermal absorption of TCB occurred from non-occluded soil. Fractional penetration of skin into the perfusate was 0.66%, absorption into dermis and other local tissues excluding stratum corneum was 2.48%, and stratum corneum absorption was 0.90%. Occlusion of the soil mixture significantly decreased dermal absorption 2-3-fold. In addition, dermal absorption from the liquid formulations (acetone, water-acetone mixture, or methylene chloride) was also significantly lower, suggesting TCB dermal absorption data from liquid formulations may considerably underestimate the risk of exposure to TCB in a soil matrix.

Qiao and Riviere (2001) performed a full mass balance in vivo study in Yorkshire-Landrace pigs after iv and dermal exposure to identical doses of 300 µg ^{14}C-TCB. For dermal exposure, TCB in acetone vehicle was applied to a 7.5 cm^2 abdominal area of three pigs and protected by a glass chamber with holes, followed by covering with a nylon sieve screening. Urine and feces were collected for 11 days, with quantitative tissue analysis and tape stripping of the TCB-exposed dermal region conducted at the end of the 11 day exposure. On average, about 70-71% of the applied dermal and iv doses were recovered. After iv dosing, a total of 60% of the dose was excreted via urinary and fecal routes with 8% of the initial dose remaining in body tissues. However, when TCB was given topically, the total excretion was only 5% but with a much larger tissue

residue of 16%. The fraction of applied dermal dose reaching the systemic circulation was estimated at 22%, with 0.85% of the applied dose in stratum corneum following tape stripping of the TCB-exposed skin.

Because of the higher tissue residue levels following dermal absorption of TCB, the researchers noted that dermal absorption of chemicals similar to TCB may be underestimated without a full mass balance analysis (Qiao and Riviere, 2001). In other words, estimating dermal absorption by comparing urinary excretion or blood AUC data with data obtained by the iv route (which represents 100% absorption) would underestimate actual TCB dermal absorption. Use of these indirect methods of absorption would provide a calculated dermal absorption of 6.3-10%.

In addition to their in vivo monkey study described above, Wester et al. (1993b) also estimated in vitro dermal absorption of PCBs through human skin from soil. The percent dose penetrating to the receptor fluid after 24 hr exposure was 0.04% for both Aroclor 1242 and Aroclor 1254. The percent dose absorbed in skin was 2.6% for Aroclor 1242 and 1.6% for Aroclor 1254. The low in vitro dermal absorption compared to their in vivo monkey study results was thought to result from tissue viability issues or solubility limits with receptor fluid. However, in vitro dermal absorption and penetration using water as the vehicle resulted in a fractional absorption of 44-46% for both PCB formulations.

The dermal absorption of purified TCB from soil was studied in rat and human skin in vitro (USEPA, 1992). The soil was comprised mostly of silt with an organic carbon content of 0.45% and a particle size range within 0.05-2 mm. The TCB concentration in the soil was 1000 ppm and soil loading was 10 mg/cm^2 for the rat skin and 6 mg/cm^2 for the human skin. After 96 hours, 7.10% of the applied dose had penetrated the human skin into the perfusate, with another 0.26% remaining in skin after washing. In comparison, total dermal absorption in rat skin was over 4-fold higher. A similar experiment was conducted with rat skin in vitro using a soil with a high organic carbon content of 11.2%. Total dermal absorption of TCB was reduced over 3-fold compared to total absorption from the low organic carbon soil.

Dermal absorption of PCBs was estimated by the disappearance method in a single volunteer exposed to a mixture of ^{13}C-labeled tetra-, penta-, hexa-, and heptachlorobiphenyls (Schmid et al., 1992). Five mg of the PCB mixture were applied to a 4 cm^2 cotton cloth in methylene chloride vehicle and dried. The cotton cloth was then applied to the tip of the forefinger or inner side of the forearm without occlusion for 8 hrs. After recovery of PCBs from the carrier and skin surface, disappearance of the remaining label suggested dermal absorption was 7 and 47% of total dose applied to finger and forearm, respectively. However, plasma concentrations of ^{13}C-label were at or below the limit of detection (10-20 pg/ml) and were not considered reliable. Application of PCBs to aluminum foil, then rubbed into the skin of the forearm for 10 min, resulted in a

fractional absorption of 8% by the disappearance method and a plasma concentration of 56.3 pg/ml. The authors suggested that the lack of measurable serum levels of PCBs was partly due to evaporative loss during exposure.

Dermal absorption of HCB in vivo and in vitro was investigated in young (33 days of age) and adult (82 days of age) female rats (Fisher et al., 1989). Young rats absorbed 3.37 times as much HCB dermally as adults in the first 6 hrs of exposure. This resulted from a lag time for penetration of about 1 hr in young and 4 hrs in adult rats. At 72 hrs in vivo dermal penetration was 35% in young and 26% in adults compared to 1.5% for young and 1.0% for adult as measured with a continuous flow in vitro system, and 2.9% for young and 1.9% for adults as measured with a static in vitro system. By 120 hrs both young and adult rats have the same cumulative dermal absorption.

F. 4.1.2 Discussion and Recommendation for a Polychlorinated Biphenyl ABS

The Wester et al. (1993b) study provided the highest fractional dermal absorption value (14%) for PCBs in soil among the in vivo experimental animal species considered most relevant for human exposures (i.e., monkey and pigs). Similar to the Wester study, Mayes et al. (2002) used Rhesus monkeys to estimate dermal absorption of PCBs, but obtained fractional absorption values of only 3-4%. Suggested reasons for the lower value include a greater proportion of highly chlorinated congeners, which reduce absorption. However, this may not be an issue because Wester got similar fractional absorption values using an Arochlor (1242) dominated by tri- and tetra-congeners, and an Arochlor (1254) dominated by penta- and hexa-congeners. Use of a soil with higher organic carbon content may have also resulted in a lower fraction absorption. Additionally, Spalt et al. (2009) notes that Mayes et al. probably exceeded monolayer coverage during the experiment, whereas Wester et al. did not.

The Wester et al. and Mayes et al. studies also used an indirect mass balance adjustment for dermal absorption by comparing excretion of dermally-applied PCBs to excretion of iv administered PCBs. Qiao and Riviere (2001) showed that this may underestimate dermal absorption up to 2- to 3-fold due to greater organ and tissue content of PCBs following dermal absorption compared to PCBs that were injected by the iv route. Thus, the highest absorption fraction estimate (14%) by Wester et al. (1993b) is recommended as the best health protective value.

Wester et al. (1993b) did not age the PCBs in soil prior to dermal application on the monkeys. However, Mayes et al. (2002) observed that aging of PCBs in soil did not reduce dermal absorption compared to freshly spiked soil.

In vitro dermal absorption studies were not considered for estimating the ABS. Comparison studies applying PCBs both in vivo and in vitro suggest that estimating dermal fractional absorption with an in vitro system would

underestimate dermal absorption obtained by in vivo methods (USEPA, 1992; Wester et al., 1993b). A reason for this underestimation may be the limited lipophilicity of the receptor fluid used with the in vitro systems.

F. 4.2 Polychlorinated Dibenzo-p-dioxins and Dibenzofurans

"Dioxin" emissions are reported as 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) equivalents. Therefore, for purposes of the Hot Spots program, all polychlorinated dibenzo-p-dioxins and dibenzofurans are considered to have the same dermal absorption characteristics as TCDD.

Recommended point estimate for dermal uptake from soil: 3%

F. 4.2.1 Studies Considered

A. Key Studies

The dermal absorption of TCDD from high organic (HOS) and low organic (LOS) soils in rats in vitro, and in human skin in vitro and rats in vivo from LOS only, was investigated during exposure intervals up to 96 hours (U.S. EPA, 1992; Roy et al., 2008). The LOS was comprised mostly of silt with an organic carbon content of 0.45% and a particle size range within 0.05-2 mm. For the in vitro studies, the TCDD concentration in the LOS was 1 ppm with soil loading of 10 mg/cm^2 on the rat skin and 6 mg/cm^2 on the human skin. After 24 hrs, 0.28% and 1.17% of the applied dose had penetrated human and rat skin, respectively, to the receptor fluid (Table F-3). Although the dose of TCDD remaining in skin was not determined at 24 hrs, the 96 hr exposure estimate in human and rat skin following skin surface wiping was 0.17 and 1.41%, respectively. The percent of applied dose reaching the receptor fluid at 96 hrs was 2.25% in human skin and 6.32% in rat skin.

The percent of dose absorbed from LOS by rats in vivo was 7.9% at 24 hrs and 16.3% at 96 hrs (Table F-3). TCDD absorbed was estimated indirectly by dividing the percent of applied dose found in the excreta by the fraction of applied dose in the excreta at the same time after i.v. administration. However, TCDD systemically absorbed at 96 hrs was also quantified in all urine, feces and tissues, resulting in 16.3% of dose absorbed. To derive an ABS for human *in vivo* uptake of TCDD from LOS (0.45% organic carbon content) and HOS (11.2% organic carbon content), USEPA (1992) applied corrections by direct ratios to account for rat in vivo, rat in vitro, and human in vitro data. For human TCDD absorption from LOS, the in vivo absorption in rat at 24 hrs was multiplied by the ratio of human to rat total absorption in vitro measured at 96 hrs. The 96 hrs data were used because this was the only measurement in which TCDD in skin was quantified. The final ABS was 2.5% (8.0% x 2.42% / 7.74%).

Table F.3. Percent Dermal Absorption of TCDD over Time from Low Organic Soil[a]

Time (hr)	Rat – in vivo	Rat – in vitro	Human in vitro
24	7.9	1.17	0.28
96	16.3	6.32	2.25
96 (Dose in skin sample after wiping)	NA[b]	1.4	0.2
96 (Total)	16.3	7.7	2.4

[a] Data from US EPA (1992) and Roy et al., 2008
[b] Not applicable

Roy et al. (2008) note that steady state conditions for the TCDD concentration in skin from LOS are reached by 24 hours for the in vitro experiments. Thus it should be reasonable to assume that the amount in the skin after 96 hours is about the same as after 24 hours. The researchers also observed that the rat in vivo percent absorbed results were about twice as high as the rat in vitro results after 96 hours. Assuming the human in vitro results would operate in a similar fashion Roy et al. obtained a human 24-hr fractional TCDD absorption rate of 0.96% (0.48% x 16.3% / 7.7%). Additionally, a fractional absorption value of 0.1% was derived for TCDD absorbed from HOS (soil with an organic content >10%).

Alternately, it may be more relevant to multiply the rat in vivo percent absorbed at 24 hours (7.9%) by the estimated in vitro rat-to-human ratio for total percent TCDD absorbed at 24 hours (0.48% / 2.75%), rather than rely on any of the results from 96 hr exposure. The resulting human 24-hr fractional TCDD absorption rate by this method is 1.4%.

B. Supporting Studies

Shu et al. (1988) applied soil-bound TCDD to the backs of rats, clipped of hair. Laboratory contaminated TCDD soil was prepared from soil obtained from Times Beach MO and determined not to contain TCDD before the experimental addition of the chemical. Environmentally contaminated soil was also obtained from Times Beach, MO and determined to contain 123 ppb TCDD after sieving through a 40-mesh screen. The organic carbon content of the soils was not specified. Soil loading was 20.8 mg soil/cm^2 skin on a total skin area of 12 cm^2. The TCDD content of the laboratory prepared soil was 10 or 100 pg/mg soil. Occlusion of the skin was minimized by the use of a perforated aluminum eye patch to cover the exposed area. Dermal exposure duration to the TCDD-laden soil was 24 hours and recovery was measured 48 hours following initiation of exposure. In some experiments, 0.5 or 2.0 percent (w/w) used crankcase oil was added to the soil before the addition of TCDD.

Following 24 hour dermal exposure + 24 hour post-exposure (total of 48 hours from initiation of exposure), the TCDD content of the liver was determined. The uptake of TCDD under the experimental protocols ranged from 0.54 ± 0.06 to 1.01 ± 0.22% and averaged 0.76 ± 0.16%. The percent uptake of TCDD in liver was not affected by the applied TCDD dose (12.5 or 125 ng/kg BW), the presence of crankcase oil in the soil, the use of soil that had been environmentally contaminated with TCDD, or by the use of haired or hairless rats.

Peak liver concentrations for TCDD administered orally and dermally were used to correct for incomplete absorption in the calculation of relative dermal absorption. The calculation is based on the assumption that the source of fecal TCDD following oral exposure is unabsorbed TCDD. The estimated relative dermal bioavailability is 1.5% from laboratory-contaminated soil and 1.6% from environmentally contaminated soil.

Diliberto et al. (1996) note that during the first 48 hours following oral exposure, TCDD in rat feces included both unabsorbed TCDD and absorbed TCDD that was excreted in bile. However the data suggest that at 48 hours, absorbed TCDD contributes only about 10% of the fecal TCDD.

Poiger and Schlatter (1980) applied radiolabeled TCDD in a soil/water paste formulation (26, 350, or 1300 ng in 14.3 mg soil/cm^2 skin) to the backs of hairless rats and measured the appearance of label in the livers. The soil (organic carbon content unspecified) was taken from the Seveso region and was TCDD-free. Measurements were taken 48 hours after the initiation of a 24 hour exposure period.

The average percentage of dose in the liver after dermal application was 0.05, 1.7, and 2.2% for the 26, 350, and 1300 ng dose groups, respectively. The authors noted that other researchers observed that 70% of total body burden of administered TCDD is found in the liver of rats. Using this estimate, the corrected dermal absorption of total applied dose is 0.07, 2.4, and 3.1% for the 26, 350, and 1300 ng dose groups, respectively. The authors also compared the liver uptake of dermally applied TCDD from a soil/water paste to the uptake from methanol, and found the soil/water paste caused a reduction in the fractional uptake (compared to methanol) of 12 percent (1.6 ng TCDD/kg BW) or 15 percent (5.8 ng/kg BW).

TCDD in acetone vehicle was applied to human skin in vitro to estimate the capacity of skin to store TCDD (Weber et al., 1991). Although TCDD did not readily penetrate the skin into the saline receptor fluid (0.03% of dose) after 16.7 hrs exposure, a major portion of the dose was found in skin. The percent of dose absorbed in skin at 16.7 hrs was 56% at a skin loading of 65 ng/cm^2, and 40% at a skin loading of 6.5 ng/cm^2.

Age may be a factor in the absorption of TCDD-like compounds. Anderson et al. (1993) applied radiolabeled TCDD in acetone (111 pmol/cm^2 applied over 1.8 cm^2) to the interscapular region of 3-, 5-, 8-, 10-, and 36-week-old rats and measured dermal absorption 72 hrs later. Dermal absorption was greatest in 3-week-old rats at 64%, decreasing to about 40% in 5-, 8-, and 10-week-old rats, and to about 22% in 36-week-old rats. Although the reason for the age-related changes in dermal absorption was not explored, the authors suggested increased lipids in skin of the young may be a factor.

F. 4.2.2 Discussion and Recommendation for a Polychlorinated Dibenzo-p-dioxin and Dibenzofuran ABS

Human skin has the capacity to store TCDD in vitro (Weber et al., 1991; Roy et al., 2008). Once absorbed in skin, lipophilic compounds such as TCDD are anticipated to be eventually absorbed into the systemic circulation. Data for another lipophilic pollutant, lindane, indicates that the chemical retained in skin will be eventually systemically absorbed (Dick et al., 1997a).

Several methods for assessing the dermal exposure data by US EPA (1992) and Roy et al. (2008) were employed above to obtain a total fractional absorption (i.e., amount that reached the bloodstream + amount retained in skin) for TCDD ABS. Since the fractional dermal absorption values presented in this document are based on 24-hr exposure, the most relevant means for estimating an ABS is to rely only on the 24-hr absorption results. The resulting human 24-hr fractional TCDD absorption rate by this method is 1.4%. Roy et al. (2008) employ a monolayer adjustment factor in their assessment, noting that the human in vitro skin test used a soil load of 6 mg/cm^2, which was greater than monolayer load by a factor of 2. Multiplying by this factor, the 24-hr TCDD fractional absorption for human skin is estimated at 2.8% for LOS, which is then rounded up to 3%.

Although both Shu et al. (1988) and Poiger and Schlatter (1980) estimated dermal absorption fractions in rats near 2%, neither study specified the organic carbon content of the TCDD-contaminated soil. The organic carbon content of soil is a major determinant for TCDD dermal absorption. At 96 hrs, USEPA (1992) noted that the ratio of TCDD absorption from low organic carbon soil (0.45% organic carbon) in rat skin measured in vitro to absorption from high organic carbon soil (11.2% organic carbon) in the same system was 7.5. Without the organic carbon content of the soil, it is difficult to compare the findings of Shu et al. (1988) and Poiger and Schlatter (1980) with that of the USEPA study.

TCDD aged in soil prior to dermal application had little effect on absorption, which is supported by the long half-life of TCDD in soil. Shu et al. (1988) observed similar dermal absorption estimates when TCDD was freshly added to soil in the lab and soil that had been environmentally contaminated with TCDD and presumably aged in the soil. In addition, soil aging of polychlorinated biphenyls (PCBs), a group of soil contaminants with some structural similarities

to TCDD, is not a significant factor for dermal absorption (Mayes et al., 2002). On the other hand, oral studies of soil-laden TCDD do indicate aging to be factor in the reduction of TCDD intestinal absorption (Poiger and Schlatter, 1980).

F. 4.3 Polycyclic Aromatic Hydrocarbons as Benzo[a]pyrene (BaP)

Recommended point estimate for dermal uptake from soil: 13%

Field studies of workers have shown that dermal absorption of PAHs may be significant. Dermal absorption of PAHs, based on the urinary excretion of 1-hydroxypyrene (1-HP), has been documented among petrochemical industry workers, including those digging in PAH-contaminated soil (Boogaard and van Sittert, 1995). Although no attempt was made to quantify the extent of absorption through dermal and inhalation routes, the results of the study strongly suggest dermal uptake is substantial and is mitigated by the use of appropriate protective clothing. Elovaara et al. (1995) compared the levels of urinary 1-HP among 6 creosote workers compared to that expected from the inhalation of the known air levels of PAHs containing $\geq$ 4 rings. Higher levels of urinary 1-HP were observed than could be accounted for solely from the inhalation route of exposure.

F. 4.3.1 Studies Considered

A. Key Study

In Wester et al. (1990b), the dermal uptake of soil-bound BaP was studied in vivo in four rhesus monkeys. The systemic absorption of soil-bound BaP was based on urinary excretion following exposure of 12 cm^2 abdominal skin to 10 ppm BaP in soil at a soil loading of 40 mg/cm^2 skin. A nonocclusive cover protected the dermal application site. Prior to sieving to approximately 180-320 µm diameter, the soil composition was 26 percent sand, 26 percent clay, and 48 percent silt with 0.9 percent organic carbon content.

Exposure duration to the chemical laden soil was 24 hours, during which time urine was collected. The cover was removed, visible soil was collected, and the skin application site was washed with soap and water. Urine was then collected for 6 additional days for a cumulative recovery period of 7 days. Incomplete excretion of BaP was corrected by the urinary excretion of BaP following intravenous (iv) administration of the PAH in acetone. The authors report a mean 24 hour dermal absorption factor of 13.2 $\pm$ 3.4 percent (Table F.4).

Radiolabeled BaP (^{14}C-BaP) was mixed with commercial gardening soil and applied in vitro onto fresh human female breast skin (obtained within 1 day of harvest) for 24 hrs by means of Bronaugh diffusion cells (Moody et al., 2007). The same amount of ^{14}C-BaP was also applied without soil to human skin samples. The soil had been sieved to <710 µm prior to spiking with BaP. The soil mixture (3.2 mg soil) was added to the diffusion cells resulting in a soil

loading of 5 mg/cm^2. At 24 hrs, the mean total percent dermal absorption including the skin depot was 14.8 and 56.4% with and without soil, respectively. The fraction of total absorbed BaP that entered the diffusion cell in 24 hrs was 7.2 and 11% with and without soil, respectively.

B. Supporting Studies

Yang et al. (1989) studied the in vivo systemic absorption in rats of BaP in soil, fortified with petroleum crude oil (1 percent (w/w)) to which ^{3}H-BaP was added. The soil, which consisted of 46 percent sand, 18 percent clay and 36 percent silt, with an organic content of 1.6 percent, was sieved to a particle size <150 µm. The final BaP level in the soil was 1 ppm and the soil loading was 9 mg/cm^2.

After 24 hours, 1.1 percent of the radioactive label was found in the rat urine and feces; no label was found in the tissues. By 96 hours (4 days) the cumulative total of radioactive label in the excreta + tissues was 9.2 percent, of which 5.8 percent was in the feces. The dermal uptake rate was estimated to be 0.2 ng/cm^2/day. Remaining BaP retained in skin at the site of application was not determined. In vitro absorption of BaP in soil was also determined in rats using a similar exposure protocol. Very good correlation was observed between the in vivo and in vitro data.

In conjunction with the in vivo dermal absorption studies in monkeys, Wester et al. (1990b) also conducted BaP dermal absorption experiments with viable human skin in vitro. Under the same soil and loading conditions of the in vivo monkey study, BaP-laden soil was applied to skin samples (dermatomed to 500 µm thickness) for 24 hrs. The percentage of applied dose in skin and in human plasma receptor fluid was 1.4 and 0.01%, respectively. When acetone was used as the vehicle under the same exposure conditions, BaP found in receptor fluid and in skin was 0.09 and 23.7% of applied dose, respectively.

Dermal absorption of ^{3}H-BaP from two different soils was determined in vitro through dermatomed pig skin cut 200 µm thick (Abdel-Rahman et al., 2002). Soil types included a sandy soil with 4.4% organic matter and a clay soil with 1.6% organic matter. Skin applications included: BaP applied as the pure compound; BaP applied immediately after the addition to each soil type (30 mg each); and pre-sterilized soils aged for three months with BaP. The chemical dose was 1.67 mg/kg and the soil loading was calculated to be 47 mg/cm^2.

Following 16 hrs of exposure, 0.2% of freshly applied BaP in sandy soil penetrated the skin to receptor fluid and 8.3% was found bound to skin. In clay soil, 0.1% of freshly applied BaP was found in the receptor fluid and 3.3% was bound to skin. In comparison, pure BaP applied to skin resulted in 0.2 and 75.8% of the dose found in receptor fluid and bound to skin, respectively. For BaP aged in either sandy or clay soil, 0.1% was found in the receptor fluid. Only 3.7 and 1.7% were bound to skin from sandy and clay soil, respectively. Aging

BaP in the soils for three months decreased total dermal adsorption by about 2-fold compared to BaP freshly applied to the soils.

Table F.4. In Vivo and In Vitro Dermal Absorption Results of Pure BaP Freshly Applied or Aged in Soils

Study	Species Treatment	Exposure time (hr)	Soil fraction (µm)	% Total absorbed fresh	% Total absorbed aged
Wester et al. 1990b	monkey in vivo	24	180-320	13.2	ND [a]
Yang et al., 1989	rat in vivo	96	<150	9.2	ND
Moody et al., 2007	human in vitro	24	<710	14.8	ND[c]
Wester et al., 1990b	human in vitro	24	180-320	1.4	ND
Abdel-Rahman et al., 2002	pig in vitro	16	unsieved	8.5[b] 3.4[c]	3.8 [b] 1.8 [c]

[a] Not determined
[b] Sandy soil
[c] Clay soil

Studies were conducted to measure in vitro absorption of BaP through human skin (previously stored frozen) from contaminated soils at manufactured gas plant (MPG) sites. These sites were impacted by PAHs in lampblack, a residue produced from the pyrolysis of oil to produce gas. Roy et al. (1998) collected nine soils from three MPG sites containing targeted PAHs at levels ranging from 10 to 2400 mg/kg. Dermal penetration rates of target PAH from the soils were determined using ^{3}H-BaP as a surrogate. Soils were sieved to <150 µm prior to analytical characterization and loaded onto skin sections at 25 mg/cm^2. Dermal absorption tests ran up to 144 hrs. The recovery of radiolabel in the receptor fluid ranged from 0.19 to 1.0%, while radiolabel absorbed in skin ranged from 0.4 to 1.0%. The highest percent of applied dose (receptor fluid + skin) from a contaminated soil was 1.9%.

Contaminated soils were collected from 7 oil-gas MPG sites in California to assess dermal absorption of BaP in vitro (Stroo et al., 2005a; Stroo et al., 2005b). The soil was sieved to <150 µm and loaded onto human skin at 10 mg/cm^2. The skin samples were dermatomed to a thickness of 350 µm. The percentage of applied dose absorbed across skin over 24 hrs ranged from 0.14 to 1.05%. The lower absorption of BaP in the lampblack samples compared to the Wester et al. (1990b) study was attributed to soil aging effects, but also to tighter binding of BaP to lampblack. Lampblack tends to bind hydrocarbons more tightly then conventional soil organic matter.

To investigate effects of soil loading and aging on PAH dermal absorption, Roy and Singh (2001) loaded PAH-spiked soil onto human skin sections at 1, 2.5, 5 and 10 mg/cm^2 following aging of the PAHs in soil up to 110 days. A field soil was sieved to <150 µm, resulting in a total organic content of 0.43%. The soil was spiked with coal tar and ^{3}H-BaP to achieve a final soil BaP concentration of 65 ppm. At soil loadings of 1 and 2.5 mg/cm^2, approximately 1% of the applied dose was in the receptor fluid at 24 hrs. The percent of applied dose absorbed decreased with increasing soil loadings of 5 and 10 mg/cm^2, respectively, indicating skin loading above monolayer coverage. In the aging experiment, the dermal bioavailability of coal-tar-derived BaP was reduced by about half by day 110 compared to the soil freshly spiked with ^{3}H-BaP.

The in vitro dermal absorption of BaP applied in acetone to full-thickness skin was compared among six mammalian species (Kao et al., 1985). The percent of applied dose permeating fresh, viable skin in 24 hrs was approximately 10% in mice, 3% in marmosets and humans, 2% in rats and rabbits, and <1% in guinea pigs. However, permeation through skin rendered non-viable by previous freezing was <1% of applied dose in all species. Permeation was accompanied by extensive first-pass metabolism of BaP in viable skin of all species. Nearly half the BaP that permeated viable human skin was attributed to BaP metabolites. In non-viable skin, essentially only unchanged BaP was detected in the receptor fluid.

PAHs have been shown to be poorly absorbed through skin from solids. No percutaneous penetration of PAHs from coal dust occurred across human skin in vitro (Sartorelli et al., 2001).

F. 4.3.2 Discussion and Recommendation for a Polycyclic Aromatic Hydrocarbon ABS

A fractional dermal absorption of 13% determined in a primate species in vivo represents a health-protective estimate of human systemic absorption of pure BaP freshly applied to an agricultural soil (Wester et al., 1990b). In support, a similar in vitro fractional absorption (14.8%) was attained by Moody et al. (2007) for 24-hr exposure of human skin to BaP-contaminated soil. The work by Wester et al. and Moody et al. were also one of the few BaP exposure studies that did not exceed monolayer soil coverage of the skin, although the coarse particle soil loadings used in the monkey study may have resulted in a lower fractional absorption.

The only other in vivo study of BaP dermal absorption from soil was in rats, in which a lower fractional absorption of 9.2% was estimated after 4-day exposure (Yang et al., 1989). Although higher organic content of the soil used could be a factor in the lower ABS in rats, the presence of petroleum crude oil (1 percent (w/w)) as a co-contaminant was also likely a factor in the lower absorption in rats compared to monkeys. Stroo et al. (2005a) note that tar in contaminated soils

tends to bind hydrocarbons more tightly than conventional soil organic matter and reduces bioavailability for dermal absorption. In addition, a soil loading of 9 mg/cm^2 exceeds monolayer coverage with soil sieved to <150 µm causing a further reduction in the percent fractional absorption.

Wester et al. (1990b) observed a roughly 10-fold lower fractional absorption of BaP in human skin in vitro compared to the human in vitro study by Moody et al. (2007). Use of a course soil fraction (180-320 µm) by Wester et al. may have reduced dermal absorption. The reduction in absorption may also be due, in part, to loss of skin viability. The Wester study used cadaver skin up to 5 days after harvest. The studies of Moody et al. obtained human skin in as little as 2-24 hrs after live donor skin harvest.

The metabolic viability of the skin samples used for in vitro studies is a factor that can affect skin permeation of BaP. Kao et al. (1985) have shown that the rate of cutaneous metabolism of BaP has a positive correlation with the permeation rate of BaP through viable skin. For example, using previously frozen human skin, as was done in some studies discussed above, renders the samples less viable and possibly much less permeable to BaP. When BaP was applied in vitro to fresh skin samples and previously frozen skin from the same individuals, a significant reduction in dermal absorption into the receiver solution was observed for the previously frozen skin (Moody et al., 2009a). However, when the skin depot was included, the difference in dermal absorption between fresh and previously frozen skin was not as pronounced.

The dermal exposure algorithm presented in Chapter 6 includes a half-life variable for BaP in soil, although it is generally assumed the half-life reflects primarily the loss of chemical due to microbial degradation. However, Adbel-Rahman et al. (2002) showed that aging of BaP in sterile soil also resulted in decreased fractional absorption in pig skin. This finding suggests BaP also shows reduced bioaccessibility over time due to partitioning into more remote sites within the soil matrix. Vigorous soil extraction procedures often used to assess soil half-life may overestimate the bioavailability of BaP because it may not be a true representation of BaP's bioaccessibility in soil for dermal absorption. Extraction techniques using human sweat or synthetic sweat would provide a more accurate estimate of the BaP half-life in soil for fractional dermal absorption studies.

F. 4.4 Hexachlorobenzene

Recommended use of default organic compound ABS estimate of 4%

F. 4.4.1 Studies Considered

No experimental data are available investigating the dermal absorption of HCB from contaminated soil. In a rat in vivo study, ^{14}C-HCB dissolved in

tetrachloroethylene was applied neat to the skin and covered with an occlusive patch after the vehicle had evaporated (Koizumi, 1991). The cumulative mean absorbed body burden, not including dosed skin directly contaminated, was 2.67% after 24 hours. Approximately 5% of the total dose remained in or on the dosed area of skin prior to washing. Washing the dosed area of skin resulted in removal of 4% of the total dose, indicating that 1% of the total dose was absorbed in the skin on which ^{14}C-HCB was directly applied.

A Monte Carlo simulation was developed to produce a probability density function for the dermal uptake fraction of HCB in soil deposited on human skin (McKone, 1991). A two-layer model was used that accounted for chemical properties, skin properties, soil properties, and exposure conditions. The resulting modeled daily dermal uptake fraction had an arithmetic mean value of 0.15 per day (24 hrs), and an arithmetic standard deviation of 0.18 per day.

F.4.4.2 Discussion and Recommendation for a Hexachlorobenzene Compound ABS

A single dermal absorption study in rats observed a 24-hr fractional absorption of 4% (rounded to nearest whole number) for the neat compound. This estimate includes HCB retained in skin at the site of application. Absorption of HCB may have increased as a result of occlusion of the exposed skin area to prevent evaporation of HCB.

A default ABS of 4% is recommended based on the rat dermal exposure study, although the chemical was applied neat to the skin. The HCB modeling study by suggests that the fractional absorption of HCB in soil may be 15%, so no adjustment was made to the ABS to account for reduced absorption due to partitioning to soil organic matter (McKone, 1991). In support, HCB is structurally similar to hexachlorocyclohexane (HCH), which has an ABS of 3%. However, the K_{ow} for HCB (log K_{ow} 5.73) is about 100 times greater than that of the HCHs, which would suggest a greater ability for absorption into skin. On the other hand, the high K_{ow} also indicates that HCB will have stronger sorption to soil organic material compared to the HCHs, which usually decreases the dermal absorption potential. Until more relevant dermal absorption studies are conducted, an ABS of 4% is recommended for HCB.

F. 4.5 Hexachlorocyclohexanes

Hexachlorocyclohexanes (HCHs) occur as eight isomers. The most common isomer is the gamma, which when purified to 99%, was sold under the trade name of lindane. Lindane was a widely used pesticide but almost all uses of lindane have been banned in the United States due to carcinogenicity concerns, high biopersistence and bioaccumulation. Dermal absorption data exist only for lindane, thus all HCH isomers are considered to have the same dermal absorption characteristics as lindane.

Recommended point estimate for dermal uptake from soil: 3%

F. 4.5.1 Studies Considered

A. Key Study

The only study located regarding dermal absorption of HCHs from soil was that of Duff and Kissel (1996) who conducted in vitro dermal absorption studies using human full-thickness skin and two lindane-contaminated soils. The organic content of the sieved sub-150 µm soils were 3.87% (sandy loam) and 0.73% (silt loam). The lindane-spiked soils were stored for up to 19 days prior to testing. No effect of aging was observed within this time frame. The studies were carried out for 24 hours with soil loading at 1, 5 or 10 mg/cm^2. The relative percent absorption decreased significantly with soil loads of 5 and 10 mg/cm^2. This was attributed to monolayer coverage of skin occurring at about 2 mg/cm^2, resulting in reduced fractional absorption at the higher soil loadings.

Results of this study showed that most of the mass of absorbed lindane was found in the skin. The average fraction of total dermal uptake found in the receptor fluid for both soils was only about 4%. Mean 24-hour total dermal absorption values (found in receptor fluid + skin) at a soil load of 1 mg/cm^2 was 1.96 and 2.35%, for low and high organic content soil, respectively. Approximately 40% of the lindane was lost to volatilization with a soil load of 1 mg/cm^2, while significantly lesser amounts were lost in the higher loading trials (less than 10% for the sandy loam soil at 10 mg/cm^2; less than 20% for the silt loam soil at 10 mg/cm^2).

B. Supporting Studies

Feldman and Maibach (1974) examined the percutaneous absorption of lindane dissolved in acetone and applied to the skin of human subjects (n = 6). Radiolabeled lindane (4 µg/cm^2) was applied to ventral forearm skin and the urinary excretion of ^{14}C was measured for 5 days after the single topical application. The skin sites were not protected and subjects were asked not to wash the area for 24 hours. Data obtained after i.v. dosing were used to correct the skin penetration data for incomplete urinary recovery. Results indicate that 9.3% (SD 3.7) of the dose was absorbed. However, when skin was occluded, the percent of absorbed dose increased dramatically to 82.1%.

In another human study, lindane was dissolved in acetone and applied to the ventral forearm of volunteers and covered with a nonocclusive patch (Dick et al., 1997a). Six hours after application approximately 80% of the applied lindane dose (120 mg lindane per ml acetone) had not been absorbed and 14% of the dose was found in the stratum corneum (measured by tape-stripping). The authors conclude that 5% of the applied dose was absorbed to the systemic

circulation by 6 hours. Although the disappearance method was used to estimate systemic absorption, measurable levels of lindane were found in the bloodstream and lindane metabolites were found in the urine. By 24 hours, tape stripping of the remaining volunteers showed the stratum corneum contained very little of the applied lindane and only about 0.01% of the dose had been lost through desquamation, suggesting that nearly all the lindane detected in the stratum corneum at 6 hours had been systemically absorbed or absorbed into deeper skin layers by 24 hrs.

F.4.5.2 Discussion and Recommendation for a Hexachlorocyclohexane ABS

Although only one study for dermal absorption of lindane from soil is available, the findings provided consistent results for a human in vitro fractional absorption range of 0.45 to 2.35% under different soil loadings and soil types (Duff and Kissel, 1996). The highest fractional absorption of 2.35% was chosen as the basis for the HCH ABS, given that the soil loading (1 mg/cm^2) used was the only one that was at or below monolayer skin coverage. An average of only 4% of the absorbed dose (approximately 0.09% of the applied dose) was found in the receptor fluid after 24 hrs. However, in vivo studies show extensive absorption of lindane into all skin layers, with continued absorption of lindane beyond the stratum corneum 6 hrs after removal of lindane from the skin surface (Dick et al., 1997a). Thus, lindane retained in skin depots should be presumed to be available for eventual systemic absorption.

Duff and Kissel (1996) noted the unexpected result that the soil with the higher organic carbon content generated a higher fractional absorption (2.35%) than the soil with low organic carbon content (1.96%) at equivalent soil loadings of 1 mg/cm^2. Increasing organic carbon content of soil generally reduces transport, and dermal absorption, of organic compounds in soil. The authors theorized that this inconsistent finding at 1 mg/cm^2 was due to inter-individual differences in skin absorption, which would not have occurred had the same skin donors been used for both soils.

To account for known effects of organic content of soil the ABS of 2.35% is rounded up, rather than down, to one significant figure for a final ABS of 3%. In support of this ABS adjustment, soil loadings of 5 and 10 mg/cm^2 from high organic content soil did reduce fractional absorption of lindane compared to lindane in soil with low organic content (Duff and Kissel, 1996). However, monolayer coverage of skin was exceeded at these higher soil loads, resulting in lower fractional absorption compared to fractional absorption at 1 mg/cm^2.

Other data available on percutaneous absorption of lindane or other HCH isomers, which are obtained from studies that use acetone or topical creams and lotions as the vehicle, are not relevant for estimating fractional absorption of lindane from soil (Franz et al., 1996). Use of topical creams and lotions as a

vehicle for lindane in dermal absorption studies is related to lindane's use as a medicine to treat scabies.

Theoretical calculations in which release from soil is not the primary limiting factor in the dermal absorption of lindane predict the percent absorbed at 55.6 to 98.5% (Bunge and Parks, 1997). The upper end of this range brackets the 82.1% absorption of applied dose observed by Feldman and Maibach (1974) when the vehicle is acetone and evaporation of lindane is limited by occlusion. However, the lower dermal absorption of lindane from soil observed by Duff and Kissel (1996) is consistent with the theory of slow soil release kinetics, in which partitioning from soil to skin is the limiting factor in dermal absorption for a number of organic compounds (Bunge and Parks, 1997). Oral bioavailability data for absorption of lindane from soil support the dermal data for absorption of lindane from soil. Soil (organic matter content of 9.8%) spiked with lindane and aged was found to have an oral bioavailability of only 7.2% in an in vitro gastrointestinal extraction test (Scott and Dean, 2005).

The dermal exposure scenario used in this document assumes that deposition of contaminated soil occurs on non-occluded skin exposed to the environment. These conditions would promote evaporation of lindane from soil on the skin, resulting in less absorption into skin than might be expected (Wester and Maibach, 1985; Duff and Kissel, 1996). A potential limitation of this ABS is if significant dermal deposition of lindane-contaminated soil occurs on skin under clothing. The situation may then become one of a reservoir for lindane in which enhanced dermal absorption occurs because of limited evaporation. However, the volatilization potential for lindane from soil also suggests that the absorption potential for lindane may be more significant when exposure is from excavated soils or from surface soils soon after the contamination event (Bunge and Parks, 1997). These various countervailing influences on dermal absorption of lindane under the exposure scenario support the assumption that the ABS will not underestimate actual dermal absorption.

F. 4.6 Diethylhexylphthalate (DEHP)

Recommend point estimate for dermal uptake from soil: 9%

F. 4.6.1 Studies Considered

A. Key Studies

No studies were located on dermal absorption of di(2-ethylhexyl)phthalate (DEHP) from soil.

Deisinger et al. (1998) estimated the migration and subsequent absorption of radiolabeled DEHP from polyvinyl chloride film into rat skin in vivo. Based on the amount of DEHP that migrated from film (505.6 mg) with 24 hr dermal exposure,

systemic absorption was estimated at 3.4% of the migrated dose. After skin washing, the residual fraction in skin at the site of dermal application was 13.8% of the migrated dose. Assuming the fraction of DEHP in skin will be eventually absorbed systemically, a maximum absorption rate of 0.24 µg/cm^2/hr was calculated.

Barber et al. (1992) carried out an in vitro DEHP dermal exposure study to compare rates of absorption through full thickness rat skin and human stratum corneum. DEHP was applied to skin samples in saline solution, and absorption expressed in terms of absorption rate after 32 hrs of exposure. Absorption through rat skin and human stratum corneum was 0.42 and 0.10 µg/cm^2/hr, respectively, indicating that DEHP more rapidly penetrated rat skin than human stratum corneum by a factor of 4.2.

Damage to the rat skin observed following exposure was implied as a possible reason for greater permeability of DEHP through rat skin. Scott et al. (1987) ~~also~~ compared absorption rates of DEHP through rat and human epidermal membranes (dermal layer removed), obtaining rates of 2.24 and 1.06 µg/cm^2/hr for rat and human skin, respectively. DEHP was applied to the skin sample in 50% v/v aqueous ethanol with exposure up to 53 hrs for rat skin and 72 hrs for human skin. Damage to rat skin, but not human skin, was also observed by Scott et al. (1987) after exposure.

B. Supporting Studies

The National Toxicology Program investigated the dermal absorption of ^{14}C-labeled DEHP in male F344 rats (Melnick et al., 1987; Elsisi et al., 1989). The labeled compound was dissolved in ethanol and applied directly to the skin (30 mg DEHP/kg body weight; n = 3 per time point) at a dose of 5-8 mg/cm^2. The ethanol was then evaporated and the site of application was covered with a perforated plastic cap. DEHP showed a very slow rate of excretion over five days, likely reflecting a slow dermal uptake process. After five days, approximately 86% of the applied dose was recovered from the skin at the site of application. However, it was not determined how much of the applied dose remained on the surface of the skin and how much was absorbed into the skin. Approximately 5% of the applied dose was recovered in urine and feces, while the amount of the label remaining in the body five days after dosing was less than 2% of the applied dose of DEHP.

Ng et al. (1992) examined dermal absorption of DEHP both in vivo and vitro in hairless guinea pigs. In an in vivo study, radiolabeled DEHP dissolved in acetone (53 µg DEHP; 34 nmols/cm^2) was applied topically on a dorsal area of the animals which was then covered with a nonocclusive patch. After 24 hours, the patch was removed and the dosing site cleaned to remove any unabsorbed compound. Absorption (estimated from urine and feces) was monitored up to 7 days post treatment. To account for incomplete excretion after the compound

was absorbed, a dose of ^{14}C-DEHP was given intramuscularly to a group of animals (n=5) and radioactivity was measured in urine and feces for up to seven days.

After 24 hours, 3% (7% after correction) of the dermally applied dose was eliminated in urine and feces. After seven days, approximately 21% (53% after correction) of the dose had been absorbed by the skin and eliminated, while another 11.3% of the dose had been skin stripped from the dose area. An additional group (n=6) of animals was given DEHP (53 μg) dermally to estimate the dose remaining in the tissues. After 7 days, ^{14}C content (% of applied dose) was as follows: urine, 18 ± 4; feces, 4 ± 1; skin wash after 24 hrs, 32 ± 10; skin patch, 13 ± 5; skin (dosed area), 5 ± 3; other tissues (liver, fat, muscle, skin), 4 ± 3%. An additional 10% was estimated to be lost to volatilization.

In the in vitro study, Ng et al. (1992) examined absorption of DEHP through viable and non-viable dermatomed guinea pig skin (200 μm sections) with 24-hr exposure. Radiolabeled DEHP was applied in 10 μl acetone at concentrations of 35.6, 153, or 313 nmol/cm^2. The percentage of dose that permeated the viable skin into the receptor fluid was 6, 2.4, and 2.5% for the low-, medium-, and high-dose groups, respectively. The percentage of dose that remained in the skin disc was 41.0, 37.5, and 36.2% for the low-, medium-, and high-dose groups, respectively. Use of nonviable skin resulted in a slightly decreased penetration of 5.0% at the applied dose of 35.6 nmol/cm^2, likely due to decreased metabolism of DEHP. There was a dose-related increase in metabolism but the total metabolites were between 0.5 and 1% of the applied dose for each dose group.

Chu et al. (1996) examined the skin reservoir effects of ^{14}C-labelled DEHP (119-529 μg/cm^2) applied on hairless guinea pigs for 24 hrs, followed by washing of the skin to remove DEHP and analysis of DEHP distribution up to 14 days post-treatment. As DEHP in the dosed skin decreased from 11.1% to 0.66% from 24-hrs to 7 days post-treatment, excreted DEHP gradually increased from 0.74 to 17.3%.

This finding provided evidence that DEHP stored in skin enters the systemic circulation, although the considerable intraspecies variation for percent of absorbed dose precluded a specific estimate of DEHP absorbed systemically after 24 hrs post-treatment. DEHP in the carcass was 1.01 and 0.92% of applied dose at 24 hrs and 7 days, respectively. By 14 days post-treatment, essentially no DEHP remained in dosed skin. Autoradiographic analysis of the dosed skin at 24 hrs revealed dense radiolabel accumulation in the epidermis and along the hair follicles, which indicated hair follicles may be a penetration pathway for DEHP.

The authors also reported that the percent absorbed at 24 hours by Ng et al. (1992) was higher than that found in this study, with nearly identical experimental

protocols. They attributed this difference to the higher doses used in the present study (10 times higher when expressed in μg/cm^2) stating that saturation might have occurred at higher doses, resulting in a lower fractional absorption.

F. 4.6.2 Discussion and Recommendation for a Diethylhexylphthalate ABS

Although two in vitro dermal absorption studies have been carried out with pure DEHP on human skin, data were not provided to determine ABS values. However, absorption rates were determined for both rat and human skin under similar exposure conditions and compared. The DEHP absorption rate for humans was 2-4 times less than that for rats (Scott et al., 1987; Barber et al., 1992).

In vivo studies in rats and guinea pigs that determined absorption of DEHP by total mass balance provide the best estimates for fractional dermal absorption in these species. Deisinger et al. (1998) used PVC film as the vehicle for transfer of DEHP to the skin of rats. Using PVC film as the vehicle will slow absorption, as DEHP requires transfer from the film before partitioning into skin can occur. This type of chemical transfer may give a closer estimate of a DEHP ABS from soil, compared to skin application of the pure compound as performed by the other studies. Including both systemic absorption and compound in skin at the site of application, a fractional dermal absorption value of 17.2% is attained from the Deisinger study. The rat-to-human absorption rate ratio of 2.1 determined by Scott et al. (1987) is then applied to give a final ABS of 9% (rounded up from 8.6%).

DEHP in the skin is included in this estimate, as Ng et al. (1992) and Chu et al. (1996) found there is significant systemic absorption of DEHP in skin up to 7 or more days after removal of DEHP from the skin surface. For this reason, the rat study by Melnick et al. (1987) was not considered in this assessment. The Melnick study did not wash DEHP off the site of skin application prior to analysis, so it is unknown how much DEHP was on or retained in the skin at the end of the 5 day exposure.

Similar to rats, Chu et al. (1996) also noted that guinea pig skin is considered generally more permeable to chemicals than human skin. Thus, it is not unexpected that the rat ABS of 17.2% is within the range of 9.5 to 18.9% (DEHP systemically absorbed + DEHP in skin) determined by the authors in guinea pigs. A limitation for this ABS is that both Ng et al. (1992) and Chu et al. (1996) reported that the percent absorbed in guinea pigs appeared to be higher at low application concentrations, although nearly identical experimental protocols were used. They attributed this difference to possible skin saturation occurring at higher doses (about 119-529 μg/cm^2), resulting in a lower fractional absorption. If saturation of DEHP in rat skin has occurred in the Deisinger et al. (1998) study, this may result in an underestimation of the fractional absorption value at soil concentrations associated with airborne releases.

Another limitation includes reliance on studies in which Cr(VI) is applied directly onto the skin (i.e., neat), rather than combined with soil, for estimation of fractional dermal absorption. Kissel (2011) has reported that fractional absorption is dependent on skin loading conditions for application of organic chemicals directly to skin. Increased skin loading of an organic chemical will result in lower fractional absorption provided complete coverage of the skin at the site of application occurs. Using PVC film as a surrogate for soil for transfer of DEHP from the film to skin is used in the estimation of the ABS, and thus reduces potential mismeasure of dermal absorption of organic compounds applied neat.

Comment [DD1]: Should say DEHP

Other limitations include ~~No~~ no data for dermal absorption of the compound bound to soil was located in the literature. In addition, no oral bioavailability studies for DEHP bound to soil could be found. Thus, no further adjustment of the ABS for absorption from a soil was applied.

F. 4.7 Dermal Absorption Fraction for 4,4' –Methylenedianiline

Recommended use of default organic compound ABS estimate of 10%.

F.4.7.1 Studies Considered

Brunmark et al. (1995) utilized a patch-test method to evaluate dermal exposure and pharmacokinetics of 4,4'-methylene dianiline (MDA) dissolved in isopropanol. Measurements of MDA were made in plasma and urine of the five human volunteers. The extent of absorption was evaluated by measuring the amount remaining in the patch after 1 hour. Determination of MDA remaining in the patch showed 25 to 29% was absorbed. The authors also describe elimination half-lives from plasma and urine.

Workers were monitored for two consecutive weeks in a fiber glass pipe factory for dermal exposure to MDA (diluted with triethyleneamine) using both cotton glove and hand wash monitoring (Brouwer et al., 1998). Urinary excretion of methylene dianiline was also evaluated. Urinary MDA levels correlated well with exposure measurements. Geometric means of daily exposure ranged from 81 to 1783 μg MDA, while 24 hour urine samples ranged from 8 to 249 μg MDA. Given that the Brunmark study identified a urinary half-life of MDA of 7 hours and that the measurements on the hands and forearms of the workers correlated strongly (0.94) with the urinary excretion of MDA, one can roughly estimate that between 10 and 14% of the MDA on the hands and forearms was absorbed by the workers.

MDA was applied in vitro to unoccluded human and rat skin for 72 hrs at a loading of 17.7-40.6 μg/cm^2 in ethanol (Hotchkiss et al., 1993). Absorption into the receptor fluid at 72 hrs was 6.1 and 13.0% of the applied dose for rat and

human skin, respectively. When the skin was occluded, the absorption at 72 hrs was significantly enhanced, reaching 13.3 and 32.9% for rat and human skin, respectively. MDA that remained in human skin at 72 hrs was 23.8 and 37.4% of the applied dose for unoccluded and occluded skin, respectively. For the rat, MDA content of the skin at 72 hrs was 57.6 and 53.1% of the applied dose for unoccluded and occluded skin, respectively. Although the data were only graphically presented, absorption through human skin into the receptor fluid at 24 hrs can be estimated at 8% of the applied dose for unoccluded skin and 20% of the applied dose for occluded skin.

The permeability of rat and human skin in vitro to MDA was assessed by Kenyon et al. (2004) over a large dose range, and the potential for skin to act as a reservoir for MDA was investigated. Dose levels of 0.01, 0.1 and 1 mg per 0.32 cm^2 skin were applied in ethanol:water (50:50) onto occluded skin for 24 hrs. No statistical difference in skin permeability was observed between rat and human skin. After 24 hrs, 27 to 52% of applied MDA had penetrated human skin to the receptor fluid. The percentage of applied MDA retained in human skin was 20%.

In another in vitro experiment, Kenyon et al. (2004) applied 0.1 mg MDA to human skin for 4 hrs, then removed excess MDA on the skin surface and the experiment continued for another 4 hrs. The cumulative absorption rate of MDA into the receptor fluid remained the same for the last 4 hrs, with only a slight decrease noted between 7 and 8 hrs. Of the total 11% of the MDA found in the skin, 5% was removed by tape stripping the stratum corneum. The remaining 6% of MDA was found in the digested skin, suggesting this amount would have been absorbed had the experiment continued longer. Considering that the lag time for appearance of MDA in receptor fluid was about 4 hrs, the authors presumed that the MDA remaining in the stratum corneum at 8 hrs would not be absorbed systemically.

No literature could be located regarding dermal absorption of MDA from soil. However, the fate of MDA added to soil has been investigated. MDA rapidly and strongly absorbs to loam soil which contained a total organic content of 1.3% (Cowen et al., 1998). However, MDA does not appear to form complexes with humic materials or form other irreversible soil binding processes. In one year, the aerobic biodegradation of MDA in silt loam soil was 40%.

F.4.7.2 Discussion and Recommendation for a 4,4' –Methylenedianiline ABS

Dermal absorption of MDA in workers is considered a more significant route of exposure than inhalation (Brouwer et al., 1998). The in vivo worker data support the in vitro human data in that dermal absorption is considerable. However, the exposure/application of MDA involved other organic solvents. The effect of solvent vehicle on absorption was not investigated.

No data could be located regarding dermal or oral absorption of MDA bound to soil. In addition, no oral bioavailability studies for MDA bound to soil could be located. Soil fate studies indicate that MDA binds strongly to soil, which would likely reduce dermal absorption considerably, and biodegrades slowly over a year's time. Thus, the default absorption value of 10% for organic compounds is recommended until soil-bound dermal studies are available.

F.5 Comparison with Other Published Dermal Absorption Factors

Two other agencies have published fractional dermal absorption estimates for some of the Hot Spots chemicals presented in this document. These values are shown in Table F.5 and are compared with the fractional dermal absorption values developed by OEHHA.

Table F.5. Published Point Estimates and Default Dermal Absorption Factors (ABS) as Percent of Selected Chemicals from Soil

CHEMICAL	ABS (percent)		
	OEHHA[a]	US EPA[b]	DTSC[c]
Inorganic chemicals			
Arsenic	6	3	3
Beryllium	~~1~~3	[d]	[e]
Cadmium	0.2	0.1	0.1
Chromium (VI)	2	[d]	[f]
Fluoride	~~1~~3	[d]	[e]
Lead	3	[d]	[e]
Mercury	4	[d]	[e]
Nickel	2	[d]	[e]
Selenium	~~1~~3	[d]	[e]
Organic chemicals			
Di(2-ethylhexyl)phthalate (DEHP)	9	[h]	[h]
Hexachlorobenzene	4	[h]	[h]
Hexachlorocyclohexanes (as lindane)	3	[h]	[h]
4,4'methylene dianiline (MDA)	10	[h]	[h]
Polychlorinated biphenyls (PCBs)	14	14	15
Polychlorinated dibenzo-p-dioxins and dibenzofurans (as TCDD)	3	3, 0.1[g]	3
Polycyclic aromatic hydrocarbons	13	13	15

[a] ABS values, as presented in this document by OEHHA. In most cases, the OEHHA ABS represent dermal absorption values based on the soil vehicle freshly spiked with the chemical contaminant and placed on skin for up to 24 hrs.

[b] (U.S. EPA, 2004)

[c] (DTSC, 1994)

[d] An ABS point estimate is not specifically listed for this chemical. For inorganics with insufficient data, USEPA (2004) states that the speciation of the compound is critical to the dermal absorption and there are too little data to extrapolate a reasonable default value.

[e] California's Department of Toxic Substances Control (DTSC, 1994) recommends using 1% as the default dermal absorption value for metals, based on Clement Associates (1988).

[f] California's Department of Toxic Substances Control (DTSC, 1994) in their Preliminary Endangerment Assessment Guidance Manual does not recommend a fractional absorption value for Cr(VI) due to lack of systemic carcinogenicity via non-inhalation routes of exposure.

[g] USEPA (2004) recommends a dermal absorption fraction from soil of 3%, or a dermal absorption fraction of 0.1% if the soil organic content is > 10%.

[h] No specific default ABS value is listed, although a default dermal absorption fraction for semivolatile organic compounds (SVOCs) of 10% as a screening method is used for the majority of SVOCs without dermal absorption fractions.

F.6. References

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Appendix G

Chemical Specific Soil Half-Lives

Appendix G

Chemical-specific Soil Half-life

The average concentration of a substance in soil (Csoil) is a function of several different variables, including deposition rate, accumulation period, mixing depth, soil bulk density, and the chemical-specific half-life, as shown in equation G-1 below:

$$Csoil = [GLC\ (Dep\text{-}rate)\ (86{,}400)\ (X)] / [Ks\ (SD)\ (BD)\ (Tt)] \quad (Eq.\ G\text{-}1)$$

where: Csoil = average soil concentration at a specific receptor location over the evaluation period (µg/kg)
GLC = ground level concentration from the air dispersion modeling (µg/m^3)
Dep-rate = vertical rate of deposition (m/sec) (see Chapter 2 for values)
86,400 = seconds per day conversion factor
X = integral function accounting for soil half-life
Ks = soil elimination time constant = 0.693/T1/2
SD = soil mixing depth = 1 cm for dermal scenario
BD = bulk density of soil = 1333 kg/m^3
Tt = total averaging time = 70 years = 25,550 days

The soil half-life is part of the integral function X determined as below:

$$X = [\{Exp\ (-Ks \times Tf) - Exp\ (-Ks \times To)\} / Ks] + Tt \quad (Eq.\ G\text{-}2)$$

where: EXP = Exponent base e = 2.72
Ks = soil elimination constant = 0.693/ T1/2
T1/2 = chemical-specific soil half-life
Tf = end of exposure duration (days); 25,500 for a 70-year exposure
T0 = beginning of exposure duration (days) = 0 days
Tt = total days of exposure period = Tf - T0 (days)

Estimating toxicant soil concentration is necessary for estimating dose from incidental soil ingestion by home raised meat, home raised produce, and dermal absorption via contact with contaminated soil.

Since the chemicals that the Hot Spots program is concerned with are emitted into the air and then subject to deposition to the soil, there are only two classes of chemicals considered. These classes are the semivolatilve organic chemicals, such as PAHs, PCBs and dioxins, and toxic metals such as hexavalent chromium, cadmium, lead, arsenic, and beryllium. Other programs that consider hazardous waste sites may be concerned with other classes of chemicals such as volatile organic solvents.

Soil extraction studies were often used to estimate soil half-life by using rigorous extraction techniques with an organic solvent (e.g., dichloromethane) to release as

much of the remaining chemical from soil as possible. The amount of chemical extracted from soil is considered the fraction that is bioaccessible for uptake. The bioaccessible fraction of a pollutant in soil, which is reduced over time by various processes, is used to estimate the soil half-life of chemicals.

An extraction procedure that mimics or parallels bioavailability is preferable for assessing exposure and risk than one whose sole virtue is the removal of the largest percentage of the compound from soil (Kelsey, 1997; Reid, 2000; Tang, 1999). This investigation suggests that mild, selective extractants may prove more useful as predictors of exposure than the methods currently used for regulatory purposes in some programs. The solvent needed for predictive purposes may vary with the pollutant and the species of concern.

Another common method to determine soil half-life of organic compounds is through mineralization, or ultimate degradation, studies. Instead of measuring the parent organic compound remaining in soil through extraction methods, mineralization studies add the radiolabeled chemical to soil, and measure the release of $^{14}CO_2$ from soil resulting from “ultimate” breakdown of the compound by microbial degradation.

Mineralization studies may be quite useful for determining the soil half-life of organic chemicals, if abiotic loss processes are minor, and if mineralization of the chemical occurs quickly once primary degradation (and presumably loss of toxicity) of the chemical takes place.

G.1 Metals

Biodegradation as such is not expected to occur with metals and other elements because of their elemental nature. However, once a metal is deposited to soil, leaching or weathering may eventually result in movement of the metal out of the system. The valence and charge of the metal in soil affects its sorption, solubility, and retention in soil. Additionally, soil pH and availability of charged sites on soil surfaces are the primary factors controlling formation of the ionic species, charged metal complexes or precipitates (US EPA, 2003).

Soil with predominately negatively charged sites is more plentiful in the United States; less than 5% of the total available charge on the soil surface is positively charged (US EPA, 2003; Fairbrother et al., 2007). For the metals that largely exist as cations in soil (beryllium, cadmium, lead, inorganic mercury and nickel), there is a greater propensity to be sorbed to soil particles. This makes them less bioavailable, but it also results in greater loading of metals into the soil because of reduced mobility and leaching.

Under most relevant scenarios, arsenic, chromium and selenium deposition to soil, typically results in formations of anionic complexes with oxygen (US EPA, 2003; Fairbrother et al., 2007). The most common forms of arsenic are arsenate (As(V)) and arsenite (As(III)), which are present in soil solution in the form of AsO_4^{3-} and AsO_3^{3-},

respectively. Similarly, selenium can be present as selenates (SeO_4^{2-}) and selenites (SeO_3^{2-}). Hexavalent chromium (Cr(VI)) can exist as chromate (CrO_4^{2-}) which is usually considered more soluble, mobile, and bioavailable than the sparingly soluble chromite (Cr(III)), which is normally present in soil as the precipitate $Cr(OH)_3$. Anionic metals generally move into pore water where they can leach out of the system faster, but are also more bioavailable.

As a default estimate, the metal content of soil is assumed to decay with a half-life of 10^8 days unless site-specific information is presented showing that soil conditions will result in the loss of soil metal content, i.e., soil aging or leaching. The 10^8 default means that significant loss or removal is not occurring within the risk assessment time frame of interest.

Some fraction of chromium (VI) will undergo reduction to the less toxic chromite (CrIII) species when deposited to soil (Bartlett, 1991; Fendorf, 2004; Stewart et al., 2003). However, oxidation reactions of chromium (III) to chromium (VI) can also occur at the same time in soil. Characterizing the reduction of chromium (VI) to chromium (III) is complex and "it is not possible to predict how chromium compounds will behave in soil until the soil environment has been adequately characterized" (Cohen et al., 1994a, citing Gochfeld and Whitmer, 1991). Several tests have been suggested for evaluating the reducing capacity of soils and may be considered in the development of site-specific information (Cohen et al., 1994a, citing Bartlett and James, 1988; Walkley and Black, 1934). These tests are described as follows:

> "(1) Total Cr(VI) Reducing Capacity. Use the Walkley-Black (1934) soil organic matter determination in which carbon oxidizable by $K_2Cr_2O_7$ is measured by titrating the Cr(VI) not reduced by a soil sample (in suspension with concentrated H_2SO_4) with $Fe(NH_4)_2(SO_4)_2$.
>
> (2) Available Reducing Capacity. Shake 2.5 cm^3 of moist soil 18 hours with 25 mL of 0.1 to 10mM chromium as $K_2Cr_2O_7$ in 10mM H_3PO_4, filter or centrifuge, and determine Cr(VI) not reduced in the extract by the s-diphenylcarbazide method.
>
> (3) Reducing Intensity. The procedure is the same as that used in (2) above except that 10mM KH_2PO_4 should be used in the matrix solution in place of H_3PO_4."

In the absence of site-specific data, the public health protective assumption is to assume that hexavalent chromium remains in the hexavalent form in the soil. In most instances this will lead to an over prediction of hexavalent chromium concentration from airborne deposition.

G.2 Organics

Organic compounds deposited in soil are subject to degradation or loss by both biotic and abiotic processes. Biotic processes include degradation by soil microorganisms. Abiotic loss of organic compounds in soil includes such processes as photochemical reactions (if on the surface of the soil) or volatilization from the soil.

For some persistent organic chemicals, such as PAHs, soil aging is the abiotic process causing the most loss. Aging is associated with a continuous diffusion and retention of compound molecules into remote and inaccessible regions within the soil matrix over time, often on the order of weeks or months, thereby occluding the compounds from abiotic and biotic processes (Northcott and Jones, 2001).

Many earlier soil half-life studies assumed that decreased soil extractability and bioavailability of chemicals with time was due to biodegradation by soil microorganisms, when, in fact, soil aging is a significant or dominant factor. Soil aging represents an abiotic loss process in which chemicals in soil become inaccessible for microbial degradation. Soil half-life of an organic compound can vary to a large extent depending on pre-treatment of soils before or after addition of the chemical to soil, the methodology used for soil extraction of the compound, and soil organic content. Other variables that can influence a soil half-life include vegetation coverage, weather and climate, and the presence of co-contaminants.

The organic carbon content of soil is often a major factor influencing the half-life of an organic compound. Increasing the organic carbon content of soils will increase sequestration and decrease bioavailability of organic chemicals. The amount of organic material in the soil is expressed as either organic carbon or organic matter. A conversion factor of 1.724 can be used to approximate the OC content of a soil that is expressed as OM (Northcott and Jones, 2001). The OC or organic matter (OM) contents of the soils used are identified in the summaries below if included in the study methodology. The OC content of the contaminated soil at a particular site can be taken into consideration if enough data are present to show that the OC content is a significant factor for the soil half-life of an individual chemical. A default assumption is available for the Hot Spots program, in which the fraction organic carbon in soil is 10%.

Considerable differences between field and laboratory half-life estimates have also been found for some organic chemicals such as PAHs (Doick et al., 2005). Pollutant fate studies are frequently performed under laboratory conditions and over short time periods. Field studies under realistic environmental conditions and protracted time frames probably represent a better estimate of the soil half-life and, therefore, carry more weight for estimating the soil half-life.

G.2.1 Creosotes

Creosotes are of concern primarily because of the polycyclic aromatic hydrocarbon content, which represent 85-90% of creosote constituents (Cerniglia, 1992). Therefore, in terms of soil half-life of this complex mixture, OEHHA recommends using the PAH half-life of 429 days for creosotes (see below).

G.2.2 Diethylhexylphthalate

Phthalates share the same basic structure of an esterified benzenedicarboxylic acid with two alkyl chains, and are chemically stable in the environment (Cartwright et al., 2000; Staples et al., 1997). Thus, the general absence of high concentrations of phthalates in the environment indicates the importance of biodegradative processes, specifically those mediated by microorganisms because higher organisms are unable to cleave phthalate's aromatic ring.

Metabolism of DEHP often results in the formation of the MEHP and phthalic acid. These metabolites retain some toxicological properties but are metabolized at a much faster rate than DEHP. Therefore, mineralization (i.e., ultimate degradation) of DEHP represents a reasonable and health protective indicator of the destruction of the phthalate's toxicological potential (Maag and Lokke, 1990). The very high Koc and Kow values for DEHP relative to other phthalates promote slower degradation in soil because a major fraction of this compound can eventually become strongly sorbed to soil organic material (i.e., soil aging) and therefore becomes much less bioavailable to soil microorganisms (Gejlsbjerg et al., 2001; Madsen et al., 1999).

Numerous microbial DEHP degradation studies are available in the literature, many of which measured degradation in unadulterated agricultural/garden soil. Only two studies were located in which DEHP soil degradation was investigated outdoors. In one study, DEHP-polluted sandy soil was mixed with compost topsoil and fertilizer, and then layered over a grass-covered plot (Maag and Lokke, 1990). White clover and grass were sown into the plot with four soil samples collected for analysis over 192 days. The depletion of extracted parent compound from soil roughly followed first-order kinetics with a half-life of 73 days.

In the other outdoor study, [^{14}C]DEHP was applied to sandy soil (pH 6.8, organic matter 0.3%) and potatoes planted the first year, followed by planting of barley during the second year (Schmitzer et al., 1988). Only 6.9% of the applied radiocarbon, mainly as DEHP, was recovered after 111 days when the potatoes were harvested. Nearly all the remaining activity, at least 92.3%, was lost to the atmosphere as $^{14}CO_2$. After 446 days when the barley was harvested, only 1.7% of the radiocarbon was found in the soil. A half-life was not determined, although assuming first order kinetics, the half-life would roughly be 30 days over the first four months of the study.

In a highly detailed laboratory study, Madsen et al. (1999) revealed that there are actually two phases in the mineralization of [^{14}C]DEHP in a sandy loam soil (pH 5.9, OC 2.5%) over a 130 day exposure - an initial phase during the first 30-60 days described well by first-order kinetics, and a late phase in which mineralization activity was much lower. This second phase was thought to represent mineralization that was increasingly regulated by strong sorption to organic matter, resulting in decreased bioavailability to soil microbes. The researchers also observed mineralization was strongly regulated by temperature, with the rate of mineralization increasing with increasing temperature. To account for diurnal swings in temperature that would occur in the field, the mean half-life over the temperature range examined (5, 10 and 20 °C) was 99 days during the initial phase and 161 days during the late phase.

A similar two-phase degradation rate for [^{14}C]DEHP was observed by Roslev et al. (1998) in a sludge-amended soil (DEHP is a common contaminant in sludge). The half-life for mineralization in a sandy loam soil (pH 5.9, organic matter 2.5%) was found to increase 2.5-fold in the late phase from 58 to 147 days.

Slow degradation of DEHP has been observed in other laboratory studies. Cartwright et al. (2000) observed that only 10% of DEHP added to a sandy clay loam soil (pH 6.25, OC 3.78%) was removed by indigenous microbes by day 70. Gejlsbjerg et al. (2001) observed an average mineralization of [^{14}C]DEHP in three Danish agricultural soils (pH 6.0-6.6, OC 2.2-3.0) to be only 13% (range = 8.46 to 21.8%) over two months. In both studies, strong sorption to soil organic matter was assumed to be the reason for slow microbial degradation.

On the other hand, rapid soil degradation of DEHP has also been observed. Kirchmann et al. (1991) determined a half-life of 20-80 days for loss of parent DEHP extracted from soil (pH 7.3, OC 1.77%), although the data suggested more of a linear disappearance of DEHP with time, rather than a first order disappearance. Shanker et al. (1985) observed a half-life of 15 days for loss of parent DEHP extracted from garden soil (pH 8.2) under a relatively high incubation temperature (30 °C).

The soil half-life of DEHP can vary greatly depending on the soil conditions, with a significant amount of the parent compound eventually being sorbed to soil organic matter for long periods and becoming recalcitrant to breakdown by soil microbes. The soil half-life of 73 days based on the field study by Maag and Lokke (1990) is used here as the default soil half-life for DEHP. Similar results were obtained in comprehensive soil mineralization studies by Madsen et al. (1999) and Roslev et al. (1998), although first order kinetics were not strictly followed over the full length of the studies.

G.2.3 Hexachlorobenzene

Hexachlorobenzene is a persistent soil contaminant that does not appear to be significantly degraded in soils by either abiotic or biodegradation processes (Isensee et al., 1976; Beall, 1976). In a simulated field experiment conducted in a greenhouse,

HCB applied to soil almost completely volatilized from the first two cm of soil after 19 months. However, only about 20% of the HCB was lost at a soil depth of 2-4 cm over 19 months. Only the parent compound was found in soil throughout the experiment suggesting HCB could be quite stable and persistent in a plowed field. It should be noted that this study used a single addition of HCB to the soil and the distribution of HCB with long-term low level (deposition) is likely to be different.

A soil half-life estimate for HCB was obtained from a controlled laboratory experiment conducted in plastic-covered pots over a period of 600 days (Beck and Hansen, 1974; Bro-Rasmussen et al., 1970). Analysis for parent compound following soil extraction showed a soil half-life for disappearance of HCB to be 969-2089 days with a mean of 4.2 years. In a similar experiment, Isensee et al. (1976) observed no loss of HCB from soil in covered beakers over a one-year period.

The data show loss of HCB from soil to be primarily by volatilization with essentially no loss due to microbial degradation. It is recommended that as a default estimate, the deposition of HCB to soil in particle form be assumed to decay with a half-life of 10^8 days, similar to the metals.

HCB accumulation in the soil from airborne sources has been shown to occur in field studies. There are a couple of mechanisms that could account for this observation. HCB could partition and bind tightly onto airborne particulate matter and then be subject to deposition. Alternatively, tight binding of gaseous HCB to soil could effectively make the soil a sink for gaseous airborne hexachlorobenzene. The studies in which hexachlorobenzene is added directly to soil establish that hexachlorobenzene below a certain depth remains in the soil, presumably bound.

G.2.3 Hexachlorocyclohexanes

The α- and γ-forms of the HCHs are the most common isomers in technical grade HCH, while the β-isomer is generally the most environmentally persistent. Similar to HCB, loss of HCH deposited on soil is expected to be primarily from volatilization, although some microbial degradation has been shown to occur with the HCHs (Spencer et al., 1988; Jury et al., 1987). HCH tilled into soil will adsorb to soil organic matter significantly reducing the potential for volatilization. HCHs can undergo dehydrochlorination by soil microbes in moist, acidic-to-neutral soils (Yule et al., 1967). Anaerobic soil conditions tend to favor faster degradation over aerobic conditions (MacRae et al., 1984).

No recent soil half-life studies for HCHs conducted in the U.S. could be located. Early field studies in the U.S. suggested a soil half-life for Lindane (γ-HCH) to be on the order of months to years (Lichtenstein and Schultz, 1959; Lichtenstein and Polivka, 1959). However, the method of detection used also included detection of relatively non-toxic degradation products of Lindane. It was also unclear if offsite atmospheric deposition of

HCHs onto the field plots was occurring, which can dramatically increase the apparent half-life of HCHs if not taken into account (Meijer et al., 2001).

Table G.1 Soil half-lives (days) for HCHs in subtropical environments of India.

	Singh et al., 1991[a]	Kaushik, 1989[a]	Srivastava & Yadav, 1977
α-HCH	55	-	-
γ-HCH	85	-	-
β-HCH	142	-	-
Technical HCH	-	23	44

[a] Half-lives are an average of cropped and uncropped soils

In an Indian field study, Kaushik (1989) monitored the loss of technical grade HCH sown into the top 15 cm of a field that remained fallow, and a field that contained plants and was watered regularly. The climate was characterized as subtropical, and the soil in both fields was sandy loam with a pH of 8.2 and an OC content of 0.8-1.0%. In the fallow field, the HCH half-life in the upper and lower 7.5-cm soil layers was 21 and 41 days, respectively, with a combined total half-life of 26 days. In the planted field, a total half-life of 20 days was recorded, with little difference in HCH loss observed between the upper and lower soil layers field.

In another Indian field study, Singh et al. (1991) determined the soil half-lives for several HCH isomers sown into the top 10 cm of cropped and uncropped sandy loam soil (pH 7.8; OC 0.63%) over a 1051 day period. Half-life values in the subtropical climate showed similar persistence in cropped and uncropped treatments. The longest half-life was observed for β-HCH (100 days cropped; 183 days uncropped) and the shortest half-life was observed for α-HCH (56.1 days cropped; 54.4 days uncropped). Another field study in India observed an average soil half-life of 44 days (range: 35 to 54 days) for a low concentration of technical grade HCH applied under cover of maize crop over three years of planting (Srivastava and Yadav, 1977).

Researchers have noted that the soil half-life for HCHs estimated in tropical climates likely underestimates the half-life for HCHs in cooler, temperate climates of the U.S. due to greater volatility, and probably higher microbial degradation, at warmer temperatures (Singh et al., 1990; Kaushik, 1989). Because temperate climate of California will tend toward lower volatility of HCHs from soil, the longer HCH half-lives determined by Singh et al. (1991) in Table G.1 are recommended for use in the “Hot Spots” program. If the HCH isomer profile in the soil is unknown, an average of the three isomer soil half-lives (94 days) can be used.

G.2.4 4,4’-Methylenedianiline

Cowen et al. (1998) investigated biodegradation of 4,4’-methylenedianiline under aerobic and anaerobic conditions using ^{14}C labeled methylenedianiline. The data

showed that, after 365 days of aerobic biodegradation in silt loam soil, 59.9% of 4,4'-methylenedianiline remained intact. Based on the aerobic biodegradation data from this study, using first-order kinetics default for dissipation of chemicals, OEHHA derived a soil half life of 455 days for 4,4'-methylenedianiline.

G.2.5 Polychlorinated Biphenyls (PCBs)

Polychlorinated biphenyls (PCBs) are a mixture of chlorinated biphenyl congeners that vary in the degree of chlorination. The degree of chlorination has a major impact on soil half life. Several different mixtures were marketed and used widely before PCBs were banned because of their toxicity, environmental persistence and bioaccumulative properties. Small amounts are generated as combustion byproducts and these emissions are subject to the Hot Spots program. The toxicity of individual congeners varies widely. For these reasons, meaningful overall soil half-life for PCBs is difficult to ascertain for situations in which PCB emissions are not speciated and the cancer potency factor for the entire mixture is applied. A half life of 940 days for Aroclor 1254 was derived by Hsieh et al. (1994). This value is used by the Department of Toxic Substances Control in CalTOX. In 2000, OEHHA proposed to use this value for all Aroclor mixtures and airborne emissions of unspeciated PCB mixtures generated from Hot Spots facilities.

Harner et al. (1995) studied four PCB congeners (28, 52, 138, 153) in air, herbage, and soil of the southern U.K. over the period 1942-1992 and observed soil half-lives ranging from 7 to 25 years (mean 18 years) (6570 days). Wania and Daly (2002) estimated soil half-lives of seven PCB congeners (8, 28, 52, 101, 153, 180, 194) ranging from 550 hours (23 days) to 1,700,000 hours (70,833 days).

Sinkkonen and Paasivirta (2000) suggested soil half-lives for eleven PCB congeners, ranging from 26,000 hours (1,083 days) to 330,000 hours (13,750 days), based on the work of Lake et al. (1992), Beurskens et al. (1993) and Brown et al. (1984).

Doick et al. (2005) studied long-term fate of two PCBs in an agricultural soil in Germany. Their observation over 152 months concluded that the soil half-lives were 10.9 years (3979 days) for PCB 28 and 11.2 years (4088 days) for PCB 52. The authors attributed the much longer soil half-lives of PCBs than estimates in other studies to length of study, field study conditions, vegetation (type and coverage), weather and climate, the presence of co contaminants and, particularly, soil type -- a high silt, high clay content, "heavy" soil with reduced water infiltration, compared with higher porosity, sandy soils.

There is great variability in soil half-lives among the PCB congeners in the above studies. The OEHHA adopted Toxicity Equivalency Factors (TEF) for individual PCB congeners (WHO97-TEF) (OEHHA 2003a); thus, it is appropriate to apply the soil half life data for these individual congeners where speciation of PCBs has been performed on facility emissions. Based on the studies above, only the data for PCB congeners with a WHO TEF (IUPAC # 77, 81, 105, 114, 118, 123, 126, 156, 157, 167, 169, 189) were

used for estimating soil half-lives in this document, unless only total PCBs are available (OEHHA 2003b).

Among the above studies, Lake et al. (1992) derived a half-life of 7.5 years for PCB 105 and 6.8 yrs for PCB 118, using the anaerobic dechlorination reaction in sediment of 15-17.5 cm deep from New Bedford Harbor, Connecticut. Beurskens et al. (1993) have estimated a half-life time of nine years for PCB 105, PCB 126, PCB 156 and PCB 169 in the anaerobic sediment. Brown et al. (1984) found the average elimination half-life for PCB 105 and PCB 118 in Hudson River sediments was 10 years. The OEHHA acknowledges that the degree of biodegradation in sediment would be different from that for a dry land scenario. Until studies in dry soil become available, the river sediment data appear to be the best choice.

Table G-2. Soil half-lives (days) for PCBs (IUPAC #) relevant to the "Hot Spots" program

Study	105	118	126	156	169	Total PCBs
Lake et al. 1992	2738	2482				
Beurskens et al., 1993	3285		3285	3285	3285	
Brown et al. 1984	3650	3650				
Arithmetic mean half-lives	3224	3066	3285	3285	3285	

The arithmetic mean half-lives for each PCB are shown at the bottom of Table G-2, and a grand mean half-life including all studied PCBs is 3229 days. This overall half-life of 3229 days is recommended as the estimated soil half-life for PCBs.

G.2.6 Polycyclic Aromatic Hydrocarbons (PAHs)

There are a variety of polycyclic aromatic hydrocarbons emitted from combustion sources. The structures vary by number and placement of fused aromatic carbon rings and functional groups on those rings. In general, it has been observed that the soil half-life increases with the increasing number of fused rings on a PAH and is correlated directly with molecular weight and K_{ow} (Northcott and Jones, 2001; Wild and Jones, 1993). The PAHs currently of toxicological concern under the "Hot Spots" program consist almost entirely of four or more rings with the prototype PAH, benzo(a)pyrene, containing five fused benzene rings. Naphthalene is carcinogenic and only has two rings but it is too volatile to be a multipathway chemical subject to deposition. Therefore, OEHHA chose to base the soil PAH half-life on those compounds with greater than three rings to avoid underestimating the accumulation of the carcinogenic PAHs in the soil.

Studies where PAHs have been added to soil have noted that those PAHs with three rings or less show significant volatilization from soil and microbial degradation, whereas PAHs with greater than three rings show little or no volatilization and slower microbial degradation (Wild and Jones, 1993; Cerniglia, 1992). In addition, a broad inverse relationship has been observed between the rate of biodegradation and the organic carbon (OC) content of the soil (Northcott and Jones, 2001; Wild and Jones, 1993). Soil half-life estimates for PAHs that currently have a potency equivalency factor (PEF) were given the greatest weight in determining a default soil half-life. Table G-3 shows the PAH half-life results from the most comprehensive studies found in the literature and a brief summary of the studies is given below.

Doick et al. (2005) conducted a field study and determined the long-term fate of ^{12}C and ^{14}C analogues of benzo[a]pyrene spiked in a cultivated agricultural soil subject to typical agricultural practices. The soil had a pH=7.2 and an organic matter content of 2.2%. Their observation over 152 months found that the soil half-life for benzo[a]pyrene was 2.7 years (982 days). These half-life values are much longer than estimates in other studies and are thought to be a result of the soil type, length of the study, use of field conditions rather than laboratory conditions, and vegetation (type and coverage).

Sewage sludge containing PAHs was applied to two agricultural soils at five dose levels (30 to 600 ton/ha) in field plots, followed by cultivation with annual crops or a perennial (willow) for up to 54 months (Oleszczuk and Baran, 2005). It was unclear from the description of the methodology if this work was an actual field study. Before addition of the sewage sludge, the soil with the annual crops had a pH=4.3 and a total organic carbon (OC) content of 1.12%. The soil with the perennials had a pH=5.8 and a total OC content of 1.21%. Analysis of 16 PAHs showed longer half-lives in the soil with the annual crops. However, the sewage sludge properties were considered as important as the type of crop used. The investigators suggested that longer half-lives of PAHs compared to other studies may have occurred due to the increased soil aging process in a soil-sludge matrix.

In a climate-controlled greenhouse experiment, sewage sludge containing PAHs was applied to four different soils to determine the soil half-life for a number of individual PAHs (Wild and Jones, 1993). The four soils ranged from a sandy clay loam agricultural soil (pH=6.6, organic carbon content, 6.04%) to a coniferous forest soil (pH=2.9, organic carbon content, 58%). Although the half-lives among 12 PAHs measured in the forest soil tended to be longest, the overall average of the sum of the PAH half-lives was not considerably higher in forest soil ($t_{1/2}$=192 d) compared to the overall average of the sum of the half-lives in the agricultural soils ($t_{1/2}$=146 d and 165 d) and a roadside soil (177 d). The authors noted that the controlled environmental conditions in the greenhouse optimize biodegradation compared to field conditions, and likely results in more rapid losses of PAHs from the soil.

Two different sandy loam soils were spiked with 14 PAHs in incubation chambers and their soil half-lives estimated over an exposure period of up to 196 days (Park et al.,

1990). One soil (Kidman sandy loam) had a pH=7.9 and an OC content of 0.5%, and the other soil (McLaurin sandy loam) had a pH=4.8 and an OC content of 1.1%. The half-lives for PAHs with PEF values ranged from 24 days to 391 days. Although the organic content and pH of the two soils differed, the biological degradation rates of the PAH compounds were not statistically different between the two soils.

In another laboratory study, Coover and Sims (1987) spiked a sandy loam agricultural soil (pH=7.9; OC content, 0.5%) with 16 PAHs and estimated the soil half-lives over a 240 day incubation period. Increasing the soil temperature was observed to increase the apparent loss of low molecular weight PAHs but had little effect on loss of five- and six-ring PAHs.

Table G-3 Soil half-lives (days) for PAHs relevant to the "Hot Spots" program

Study	Ch	BaA	BaP	BbF	BkF	DahA	DaiP	Ind	DaA
Coover & Sims, 1987[a]	1000	430	290	610	1400	750		730	
Park et al., 1990[b]	379	212	269	253		391	297	289	24
Wild & Jones, 1993[c]	215	215	211	202	301				
Doick et al., 2005			982						
Arithmetic mean half-lives	531	286	438	355	851	571	297	510	24

Abbreviations: Ch, chrysene; BaA, benz[a]anthracene; BaP, benzo[a]pyrene; BbF, benzo[b]fluoranthene; BkF, benzo[k]fluoranthene; DahA, dibenz[a,h]anthracene; DaiP, dibenzo[a,i]pyrene; Ind, Indeno[1,2,3-c,d]pyrene; DaA, 7,12-Dimethylbenz [a] anthracene

[a] Environmental temperature held at 20C

[b] Average half-life values for two sandy loam soils

[c] Average half-life values for four different soils. Ch and BaA co-eluted; the $t_{1/2}$ is for both PAHs combined

The arithmetic mean half-lives for each PAH are shown at the bottom of Table G.3, and a grand mean half-life including all PAHs is 429 days. Greater differences in PAH half-lives are seen between studies rather than within studies. One possible reason is that longer half-lives are attained from field studies (Doick et al., 2005) compared to laboratory studies (Coover & Sims, 1987; Park et al., 1990; Wild & Jones, 1993).

However, the limited number of field studies makes it difficult to confirm this assumption. The overall PAH half-life of 429 days is recommended until further field studies are conducted.

G.2.7 Polychlorinated Dibenzo-p-dioxins and Dibenzofurans (PCDD/F)

The prototype compound and most potent of the dioxin and furan family of compounds is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). The degree and placement of chlorination affects both the toxicity and soil half life of dioxins and furans. Sampling of 32 sites in Seveso, Italy, produced an initial calculated regression half-life of one year (365 days) (Di Domenico et al., 1980). Experimental application of TCDD to two different soil types (loamy sand and silty clay loam) for 350 days produced calculated half-life values ranging from 394 to 708 days (Kearney et al., 1972; Kearney et al., 1973). Soil half-life estimates ranging from 10 to 12 years (3650-4380 days) were reported based upon experimental measured soil concentrations of TCDD from a contaminated site at an Air Force base in Florida (Young, 1981). Soil half-life estimates of 10 to 100 years (3650-36500 days) were reported, depending on the depth of the contamination, with deeper soil having reduced biodegradation rates (Nauman and Schaum, 1987). An estimated soil half-life of 3609 days has also been reported (calculated from a soil reaction rate constant of 8×10^{-6} hr^{-1}) (Mackay et al., 1985).

Several other half-life estimates have also been identified and summarized (Cohen et al., 1994b). Soil samples showing loss of TCDD content by volatilization produced estimated half-lives of 7-24 days (Nash and Beall, 1980). TCDD measured in soils from the contaminated site in Seveso, Italy, produced a half-life estimate of 9.1 years (3322 days) (Cerlesi et al., 1989). A half-life estimate of 3 days was made based on loss of TCDD content from soil by both photodecomposition and volatilization (Di Domenico et al., 1982).

McLachlan et al. (1996) studied PCDD/F persistence in a sludge-amended soil sample with presence of PCDD/Fs from 1968 to 1990. Half-lives for these PCDD/Fs in the sludge-amended soil after 1972 were of the order of 20 years (7300 days).

There is great variability in soil half-lives among the PCDD/F congeners between the above studies. Soil half-life estimates for PCDD/Fs that currently have a toxicity equivalency factor (TEF) were given the greatest weight in determining a default soil half-life, where speciation of PCDD/Fs has been performed on facility emissions, unless only total PCDD/Fs are available (OEHHA 2003). Table G-4 shows the PCDD/F half-life results from the study (Kjeller and Rappe, 1995) found in the literature which speciated PCDD/F congeners.

Table G-4 Soil half-lives (days) for PCDD/Fs relevant to the "Hot Spots" program

Compound	TEF_{WHO-97}	Half-life (days) from Kjeller and Rappe (1995)
PCDDs		
2378-TCDD	1	37,500
12378-PeCDD	1	42,000
123478-HxCDD	0.1	100,000
123789-HxCDD	0.1	29,200
123678-HxCDD	0.1	23,000
1234678-HpCDD	0.01	37,500
12346789-OCDD	0.0001	54,200
PCDFs		
2378-TCDF	0.1	23,000
12378-PeCDF	0.05	18,750
23478-PeCDF	0.5	23,000
123478-HxCDF	0.1	25,000
123789-HxCDF	0.1	20,800
123678-HxCDF	0.1	29,200
234678-HxCDF	0.1	18,750
1234678-HpCDF	0.01	14,600
1234789-HpCDF	0.01	12,500
12346789-OCDF	0.0001	10,400
Arithmetic mean half-lives		30,600

The arithmetic mean of the suggested values from ten studies (6,986 days) cited above is recommended as the estimated soil half-life of TCDD/Fs if the facility is reporting emissions of total dioxins and furans.

G.2.8 Summary

The chemical-specific soil half-lives for each chemical are summarized as Table G-5 below.

TableG-5. Summary of Soil Half-life Values (Days).

Compound	Soil Half-life (days)
Arsenic	1.0 E+08
Beryllium	1.0 E+08
Cadmium	1.0 E+08
Chromium	1.0 E+08
Diethylhexylphthalate	1.5 E+01
Hexachlorobenzene	1.0 E+08
Hexachlorocyclohexanes	9.4 E+01
Lead	1.0 E+08
Mercury	1.0 E+08
4,4'-methylenedianiline	4.6 E+02
PAHs	4.3 E+02
PCBs	3.2 E+03
PCDD/F	7.0 E+03

For a chemical with individual congeners, such as PCBs, PAHs, PCDD/Fs, only the grand average was presented in Table G-5. When speciation of these chemicals has been performed on facility emissions, soil half-life data for individual congeners are summarized in Table G-2 (PCBs), Table G-3 (PAHs) and Table G-4 (PCDD/Fs).

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Appendix H

Root Uptake Factors

H.1 Introduction

Root uptake factors for crops have been estimated for toxic metals in the "Hot Spots" program. These toxic metals are subject to soil deposition and subsequent uptake by the roots of home raised produce. A root uptake factor is necessary to estimate a concentration in the plant from the concentration in the soil. An estimate of produce consumption can be applied to estimate dose to the residential receptor (Chapter 7). The soil-to-plant uptake factor (UF) is the ratio of the fresh weight contaminant concentration in the edible plant or plant part over the total concentration of the contaminant in wet weight soil:

$$UF = C_{f.w.plant} / C_{wet.w.\ soil} \qquad \text{(Eq. H-1)}$$

where: $C_{f.w.plant}$ = fresh weight concentration in the plant (mg/kg)
$C_{wet.w.\ soil}$ = wet weight concentration in soil (mg/kg)

In the last 25 years, a large number of studies have been published that investigated metal concentrations in edible plants grown in contaminated soils. Although most of these studies did not calculate the UF, data were often presented from which a UF could be calculated. OEHHA assembled the data from these studies into a database from which basic statistical analyses for chemical UFs were determined. The volume of studies that could be included in the database is quite large for some inorganic metals, with new studies frequently published. Our database is not an exhaustive compilation of all plant uptake studies published, however, enough data were found to reasonably estimate default UFs for most of the toxic metals and metalloids of concern.

The UFs calculated by OEHHA are based on the total metal concentration in soil and reflect the fact that most crop uptake studies estimate total metal soil concentration, usually by extraction with strong or moderately strong acids (e.g., 4 N sulfuric acid). A smaller body of uptake studies uses various mild soil extraction processes (e.g., extraction with diethyltriaminopentaacetic acid) to estimate plant bioaccessible metal concentrations in soil. Once more studies become available using an established method for estimating bioaccessible metals in contaminated soil, OEHHA may also consider developing an algorithm that incorporates a bioaccessible metal uptake factor.

The ability for crops to accumulate and translocate toxic inorganic metals and metalloids to edible parts depends to a large extent on soil and climatic factors, plant genotype and agronomic management (McLaughlin et al., 1999). In order to be most applicable to Hot Spots risk analysis, a set of criteria was applied for the selection of data used in developing soil-to-plant uptake factors.

Data used to determine root uptake factors were limited to studies that estimated contaminant concentrations in edible portions of crops raised and harvested at maturity for human consumption. Crops that are commonly grown in backyard gardens in California were considered most relevant. For example, plant uptake studies in crops grown in tropical climates were not included in the database. Grain crops such as

wheat and rice were also not included in the database because these crops are unlikely to be grown in backyard gardens. In most field studies background soil contaminant levels were unknown or not presented. However, field studies were included in the database if the study indicated that the soil was contaminated due to human causes, or that the soil contaminant concentration was considered above background levels.

Another data selection factor was soil pH because soil pH is a major influence on root uptake. Most agricultural soils in California are near neutral, with a geometric mean pH=7.2 (Holmgren et al., 1993). The range of pHs for most agricultural soils in California are roughly estimated at between 5.5 and 7.6. Thus, plant uptake studies that investigated soils with pH values within this range were considered most useful for estimating soil-to-crop uptake factors. Acidic soils tend to increase the bioavailability of divalent cationic metals such as cadmium, lead, and mercury. UFs based on acidic soils may overestimate metal uptake from pH neutral soils.

A distinction is made in the database for contaminant source between freshly added inorganic salts and other forms of the chemicals. In general, fresh addition of metal salts to soil in laboratory experiments will represent the most available form of the metal to plants. UFs developed from these studies likely represent an upper limit for plant accumulation. Where possible, UFs were calculated based on field studies that estimated plant uptake due to human-caused contamination of soils. These sources primarily included mine waste, smelter deposits, vehicle and other urban emissions, other industrial sources, wastewater effluent, compost, fertilizer, dredged material, sewage sludge, fly ash and flue dust. Ideally, UFs would be based on airborne deposition of contaminants due to emissions from nearby industrial facilities. However, uptake data from these sources were often very limited.

Most of the plant uptake studies summarized in the database presented their contaminant concentration results on a dry weight basis for both the plants and the soil. However, the soil-to-plant UF in Eq. 7.6 (Chapter 7) is expressed as a ratio of fresh weight crop concentration per wet weight soil concentration. To adjust the soil-to-plant UFs to a fresh weight crop basis, dry-to-wet weight fractions of edible portions of crops were applied using literature sources containing water content data of raw fruits and vegetables (Watt and Merrill, 1975; Baes et al., 1984; USDA, 2009). A default value of 0.8 was applied to all UFs for the dry-to-wet weight adjustment of soil, unless water content data of soil was presented in the study (Clement Associates, 1988).

As a result, two types of soil-to-plant UFs can be generated for each metal contaminant: one based on the dry weight plant over dry weight soil, and the other based on fresh weight plant over wet weight soil. A UF based on dry weights of plant and soil may be beneficial because the ratio avoids the naturally wide variations in water content of the crops and the soil. On the other hand, estimates of fruit and vegetable consumption are based on fresh weight values for the crops, which were grown in irrigated soils. This type of UF is most applicable for contaminant exposure via the crop consumption pathway (Eq. 7.6).

Finally, some studies also presented uptake data for reference soils. This information was also entered into the database to estimate crop uptake based on control soils as well as crop uptake specifically due to deposited contaminants (i.e., contaminated soil minus control soil metal concentration). Metals of concern naturally present in soils may be largely present in the mineral fraction of the soil and not available for uptake by plants. However, it may be beneficial to know what the background soil-to-plant UF is for toxic metals to estimate the impact of anthropogenic sources of the same metals is on the soils and plants.

The database of the studies used in the analysis is presented at the end of this appendix. Studies were grouped according to each metal/metalloid for comparison purposes.

H.2 Arsenic

Arsenic can be present in well-drained soil as $H_2AsO_4^{-1}$ if the soil is acidic or as $HAsO_4^{-2}$ if the soil is alkaline (Bhumbla and Keefer, 1994). Arsenite (As(III)), the reduced state of inorganic arsenic, is a toxic pollutant in natural environments. It is much more toxic and more soluble and mobile in soil than the oxidized state of inorganic arsenic, arsenate (As(V)). Under flooded conditions, As(III) would dominate, whereas aerobic conditions would favor the oxidation of As(III) to As(V). Arsenic accumulates in roots of plants grown on soils contaminated by arsenic pesticides. However, arsenic is not readily translocated to above-ground parts.

Although background mean levels of arsenic in U.S. agricultural soils could not be located, a review by Wiersma et al. (1986) showed mean levels of arsenic in European and Canadian agricultural soils to be in the range of 5 to 12 mg/kg dry soil. Kloke et al. (1984) reports that the range of arsenic in arable land to be 0.1 to 20 mg/kg dry soil. The typical dry weight concentration of arsenic in plants has been listed as 0.1 to 5 mg/kg (Vecera et al., 1999). In this document, all crops grown in As-polluted soils had an overall average dry weight arsenic concentration of about 2.5 mg/kg, which is within the range of typical plant concentrations.

Table H.1 Distribution Parameters for Arsenic Fresh Weight Soil-to-plant Uptake Factors

	Leafy	**Exposed**	**Protected**	**Root**
n	27	22	8	17
minimum	0.000275	0.0000538	0.000115	0.000338
maximum	0.055	0.132	0.27	0.045
mean	0.00983	0.0158	0.066	0.00828
median	0.00531	0.00138	0.032	0.00399
90^{th} percentile	0.0257	0.0403	0.19	0.0236
95^{th} percentile	0.0481	0.0674	0.23	0.0361

It was observed that lower UFs were recorded in plants growing in high As-polluted soils compared to plants growing in low-level As-polluted soils. This finding, in part, led to the large range in UF values shown in Table H.1 for some types of crops. For example, in soils with low-level As contamination of < 12 mg/kg, a UF of 0.01 was calculated for both exposed and leafy crops. In exposed and leafy crops grown in soils with >12 to 745 mg/kg As (mean: 343 mg/kg), calculated UFs were 0.0002 and 0.002, respectively. This seems to suggest that many crops have the ability to resist uptake, or have a high excretion rate, of excessive amounts of As in highly polluted soils. The crop UFs in Table H.1 are based on the arithmetic mean value for low- to high-level As polluted soils.

H.2 Beryllium

Very little data could be found regarding plant uptake of beryllium from the soil. Measurable amounts of beryllium in plants are rarely observed and the toxicity of this metal to plants is reported to be high (Shacklette et al., 1978; Baes et al., 1984). Kloke et al. (1984) estimates that a general dry weight plant/soil transfer coefficient for Be is in the range of 0.01 - 0.1, similar to that found for lead and mercury.

Single soil-to-plant data points from Baes et al. (1984) for leafy and protected crops were used in Table 7-6 to represent these particular crop types. These were the only UFs that could be located in the literature. Due to expected similarities in soil-to-plant transfer, the lead UFs for root and exposed crops were used to represent the root and exposed UFs for beryllium.

H.3 Cadmium

Cadmium has the most extensive literature on root uptake of any of the toxic metals Compared to Pb, Cd is readily taken up by plants, but unlike the other heavy metals, Cd is not phytotoxic at low plant concentrations that pose a concern to human health (McLaughlin et al., 1999). Cadmium exists in solution mostly as the divalent cation, Cd^{2+}. Plant uptake of Cd is governed by a number of factors that include soil pH, organic matter, cation exchange capacity, clay type and amount, hydrous metal oxides, carbonates, and other inorganic compounds (Mahler et al., 1987; McLaughlin et al., 1996). Acidic soils, and soils with lower clay and humus content will increase availability of Cd to plants.

The mean concentration of Cd in uncontaminated U.S. agricultural soils is 0.27 mg/kg d.w., with 5^{th} and 95^{th} percentiles of 0.036 and 0.78 mg/kg d.w., respectively (Holmgren et al., 1993). The mean concentration of Cd for field-contaminated soils reviewed in this document was about 8 to 9 mg/kg d.w., with a range of 0.16 to 106.5 mg/kg d.w. Typical dry weight levels of Cd in plants are expected to be between 0.1 and 1 mg/kg (Vecera et al., 1999). In this document, the overall Cd concentration in crops grown in Cd-polluted soil was about 6 mg/kg.

Figure H.1. Cumulative distribution of the leafy crop UFs for cadmium from field studies in the literature (n=73, skewness = 3.05, kurtosis = 9.09)

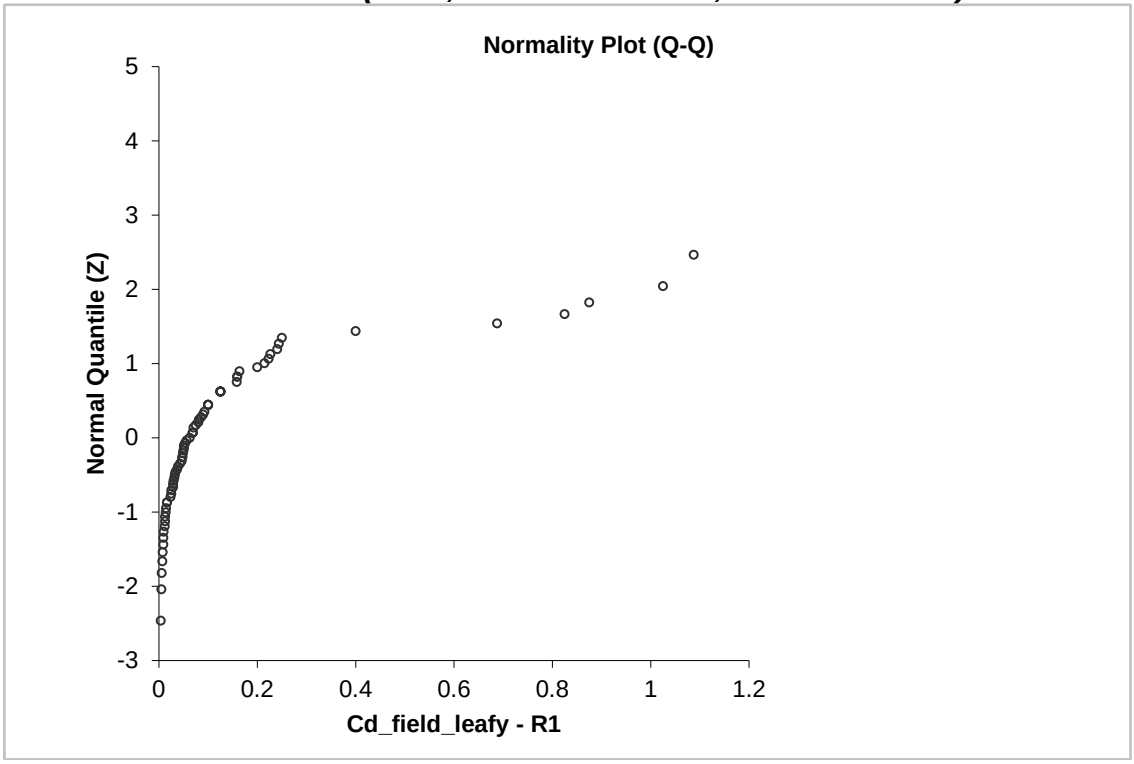

Table H.2 presents the UF distributions from field data only. UFs calculated from laboratory studies in which Cd salts were added to soils were not included in Table H.2, although there are a considerable number of these types of studies. Comparison of UFs calculated from field and Cd salt studies showed significantly greater UFs were obtained in crops grown in Cd salt-contaminated soil. For example, the mean leafy UF from Cd salt studies was 0.5 (n=27), which was significantly greater ($p<0.0001$) than the leafy UF of 0.1 based on field studies (Table H.2). The field studies were chosen to calculate the UFs because they are likely more relevant for "Hot Spots" facility soil contamination.

Table H.2: Percentile Distribution for Cadmium Fresh Weight Soil-to-plant Uptake Factors

	Leafy	**Exposed**	**Protected**	**Root**
n	81	41	27	62
minimum	0.00375	0.0001	0.0002	0.00113
maximum	1.09	0.148	0.0688	0.913
mean	0.139	0.0216	0.0134	0.0683
median	0.0688	0.008	0.0064	0.0244
90^{th} percentile	0.244	0.0541	0.0294	0.124
95^{th} percentile	0.688	0.0863	0.0552	0.172

H.4 Chromium VI

Exposure to hexavalent chromium (Cr(VI)) as a contaminant in soil has been a contentious and complex risk assessment issue that has never been satisfactorily resolved. In both industrial and environmental situations Cr(III) and Cr(VI) can inter-convert, with reduction of Cr(VI) to Cr(III) generally being favored in most soils and sediments. Rapid oxidation of a portion of Cr(III) salts or hydroxides added to almost any soil with a pH above 5 was found to occur readily, provided the soil sample was fresh and kept moist and directly from the field (Bartlett and James, 1988). However, oxidation of Cr(III) to Cr(VI) in field soils is slow compared to well mixed soils in laboratory studies, and given opportunities for its reduction, accumulated Cr(VI) from inorganic sources may rarely be measurable.

Cr(VI) added to soils may be reduced, or absorbed, or may remain in solution depending on the organic matter content, pH, and texture of the soil (Cary, 1982). In neutral to basic soil, chromium will be more available to growing plants than in acidic soil probably due to the increased stability and presence of Cr(VI) in the basic pH range. For example, when Cr(VI) was added to near-neutral pH soil (6.65) under field conditions, most of the Cr(VI) was extracted from the soil unchanged three weeks later (Bloomfield and Pruden, 1980). Under the same field conditions, most of the added Cr(VI) to an acidic soil (pH 4.20) was reduced three weeks later. These results suggest that in some neutral pH agricultural soils, such as those found in California, constant deposition of Cr(VI) may result in accumulation of Cr(VI) in the soil and ground water.

As a soluble anion, Cr(VI) readily penetrates cell membranes, whereas Cr(III) is soluble at biological pHs only when organically complexed in low molecular weight organic complexes and, therefore, soil forms probably do not penetrate membranes (Bartlett and James, 1988). The difficulty for risk assessors is attempting to estimate what proportion of chromium deposited as Cr(VI) to soil will be available for plant uptake, presumably as Cr(VI). This problem is compounded by the difficulty of estimating the actual speciation of chromium in biological tissues during analysis. As a result, most studies only measure total chromium contents of plant parts.

Cr(III) in soil probably does not penetrate plant cell membranes as such, but is thought to undergo enhanced solubility in soil due to organic acids exuded by roots (James and Bartlett, 1984; Bartlett and James, 1988). This in turn leads to an increased oxidation of Cr(III) to Cr(VI) by soil manganese oxides. The oxidation of Cr(III) to anionic Cr(VI) enables its absorption by the roots. However, once absorbed by root tissues, it appears that most of the Cr(VI) is reduced again to Cr(III) and retained by the roots in a tightly bound or insoluble form or in a soluble complex (e.g., trioxalato chromate(III)) that is not translocated to the above-ground plant parts.

Evidence for the low translocation of chromium from roots has been observed by Lahouti (1979), in which crops that accumulated chromium from nutrient solutions labeled with either ^{51}Cr(III) or ^{51}Cr(VI) retained about 98% of the elements in the roots. Of nine species of crops examined, the roots supplied with ^{51}Cr(III) contained more

chromium than those supplied with $^{51}Cr(VI)$, but chromium added as $^{51}Cr(VI)$ was slightly better translocated to the shoots. In another study, onion plants were grown in soil after equivalent doses (total dose not provided) of either Cr(III) or Cr(VI) added to the soil (Srivastava et al., 1994). At the lower levels that did not injure the onion plants, the chromium concentration in the plants with Cr(VI) added to soil was only marginally higher than those with Cr(III) added to soil, with most of the chromium retained in the roots and bulb.

This finding seems to suggest that much of the chromium, either added as Cr(VI) or Cr(III), had reached an equilibrium in the soil prior to uptake by the roots.Field studies in which soils were contaminated by anthropogenic sources of Cr(VI) were difficult to come by. Soils contaminated with chromium, generally from sewage sludge, tannery waste, inorganic native chromium in mine waste, are mainly present as Cr(III). Often, the contaminated soils did not exhibit concentrations above the range of typical soil chromium levels of 2 to 50 mg/kg (Kloke et al., 1984), and no chromium control level was provided in the study. Quantitative data for plant uptake of chromium added as Cr(VI) in greenhouse studies are also limited. Cary et al. (1977a, 1977b) added Cr(VI) as K_2CrO_4 to soil over the first 29-40 days after seeding several crop varieties in pots, and then harvested the crops at maturity 70-110 days after seeding. From these data, leafy, exposed and protected crop UFs for total chromium were estimated (Table H.3). For the root UF, it was observed that roughly 10% of the chromium added as Cr(VI) to soil was incorporated in the above-ground plant parts, with the remainder incorporated into roots and bulbs (Srivastava et al., 1994). The difference between above-ground and root chromium was also reflected by a 10-fold greater concentration of chromium in roots compared to above-ground plant parts. Thus, the root UF is 10-fold greater than the leafy UF. It is currently unknown what proportion of chromium as Cr(VI) will be found in edible crops following absorption and translocation from the roots (Cary, 1982; Kimbrough et al., 1999). Bartlett and James (1988) surmised that if Cr(III) were to be translocated to above-ground plant parts, it is not unreasonable to think that if it enters the chloroplasts it might be oxidized to Cr(VI) in the powerful oxidative environment within the chloroplasts where water is oxidized to O^{2-}. Skeffington (1976) showed that 0.5% of the Cr(III) mixed with ground fresh barley roots was oxidized to Cr(VI). These data would suggest that a fraction of the chromium in roots is present as Cr(VI). Until further characterization of the form of chromium found in edible crops is determined, the health protective assumption is that the chromium found in crops due to root uptake is in the form of Cr(VI).

Table H.3: Crop uptake factors for total chromium, added originally as chromium(VI) to the soil[a]

	Leafy	Exposed	Protected	Root
N	3	1	3	-[b]
Minimum	0.18	-	0.0034	-
Maximum	0.42	-	0.19	-
Mean	0.3	0.02	0.07	3

[a] Data were too limited to determine percentiles.
[b] No quantitative data could be found for a root UF. The general finding that root levels of chromium are 10-fold greater than above-ground plant parts was to devise a root UF.

H.5 Fluoride

Fluoride (F) is strongly sorbed to soil when added as a salt, much stronger than the other halide salts of iodine, bromine and chlorine (Sheppard et al., 1993). The generally low soluble F in most soils coupled with the fact that the root endodermis acts as a barrier means that transport from root to shoot will be limited (Davison, 1982). The lack of soil-to-plant field data for fluoride resulted in a reliance on laboratory studies which added fluoride salts to the soils. The resulting UFs are shown in Table H.4.

The most important F exposure route for plants is uptake via airborne deposition of soluble fluorides of HF and particulate fluoride salts on leaf surfaces. Fluoride that deposits on leaf surfaces can be taken up through stomata of leaves once it deposits on the surface. Uptake of F into plant leaves occurs by passive permeation of the undissociated HF molecule across the plasmalemma (Kronberger, 1987). Thus, HF behaves like a weak acid (pKa = 3.4) when dissolved in water, where the ionic species becomes trapped within membrane-surrounded compartments after nonionic diffusion. Little fluoride moves downward in plants to roots, from leaf to leaf or from leaves to fruits. Assessing fluoride UFs for leafy crops near airborne industrial emissions of fluoride compounds may eventually require a different algorithm to estimate airborne fluoride accumulation in leafy crops.

Tea plants (*Camellia sinensis*) are known to accumulate high concentrations of F in their leaves from soil containing elevated levels of F, resulting in considerable amounts of F in tea beverages (Davison, 1983). However, it is not known if significant cultivation of tea plants occurs in California. There is also some evidence spinach can accumulate F from soil to a greater degree than other leafy crops (Kumpulainen and Koivistoinen, 1977). The maximum fluoride UF for leafy crops shown in Table H.4 is for spinach.

Table H.4: Fresh weight soil-to-plant uptake factors for fluoride[a]

	Leafy	Exposed	Protected	Root
N	5	-[b]	1	2
Minimum	0.0006	-	-	0.003
Maximum	0.16	-	-	0.014
Mean	0.036	0.004	0.004	0.009

[a] Data were too limited to determine percentiles.
[b] No quantitative data could be found for an exposed crop UF, so the protected crop UF was used

H.6 Lead

Deposited lead (Pb) is strongly retained by most soils, resulting in lower plant concentrations (and lower UFs) relative to more bioaccessible metals such as cadmium and nickel (McLaughlin et al., 1999). Because of the usually low soil-to-root uptake, the above-ground plant parts are likely predominantly contaminated by airborne deposition of lead-containing dust or aerosols onto the plant surface (McBride, 1998). This finding emphasizes the importance of selecting studies in which the leafy plant samples are thoroughly washed prior to assessing root uptake and translocation of lead. Because inorganic lead most often exists as a divalent cation, maintaining alkaline soil conditions will reduce lead mobility in soil, while acidic soil conditions has been shown in some cases to increase soil mobility and uptake of lead through plant roots.

The mean concentration of Pb in uncontaminated U.S. agricultural soils is 12.3 mg/kg, with 5th and 95th percentiles of 4.0 and 23.0 mg/kg, respectively (Holmgren et al., 1993). The range of Pb concentrations in field-contaminated soils reviewed in this document was large, ranging from 11 mg/kg dry soil to nearly 5500 mg/kg dry soil. Typical dry weight concentrations of Pb in plants are reported to be 0.1 to 5 mg/kg (Vecera et al., 1999), whereas the overall average Pb concentration in crops grown in Pb-polluted soil reviewed in this document was about 9.5 mg/kg.

Table H.5: Percentile distribution for lead fresh weight soil-to-plant uptake factors

	Leafy	**Exposed**	**Protected**	**Root**
n	77	38	24	57
minimum	0.0000375	0.00002	0.000075	0.0000425
maximum	0.0413	0.0475	0.0278	0.0375
mean	0.00770	0.00693	0.00282	0.00403
median	0.00298	0.00228	0.000912	0.00125
90th percentile	0.0248	0.0214	0.00465	0.00962
95th percentile	0.0308	0.0406	0.00711	0.015

H.7 Mercury

Determining the crop uptake of inorganic mercury (Hg) from soil can be problematic. (Caille et al., 2005) found that following application of radiolabeled $^{203}HgCl_2$ to sediment in a pot experiment, 33-73% of the leaf content in cabbage, rapeseed and pasture grass was due to volatilized Hg absorbed into the leaves. Presumably, the applied inorganic Hg^{2+} was emitted from the soil after reduction to Hg^0 in the soil whereupon it was absorbed by the leaves. Lindberg et al. (1979) observed the same phenomena in alfalfa grown in a chamber, in that above-ground plant parts primarily absorbed Hg vapor released from the soil originally contaminated with mercury mine waste including cinnabar (mercury(II) sulfide). However, the root levels of mercury were determined by direct uptake from contaminated soil and reflected the total Hg concentrations in the soil. Significantly, any Hg vapor emitted by a facility could also be absorbed directly onto leafy crops.

Nearly all studies examined by OEHHA for crop Hg uptake from soil measured total Hg content and did not account for potential volatilization of elemental Hg from soil. Therefore, the soil-to-plant UF for mercury in above-ground plant parts (primarily leafy) includes both root uptake from soil and leaf uptake through volatilization from soil. It is unclear what portion of Hg oxidizes to inorganic Hg once absorbed by leaves, although mercury in food stuffs are mainly in the inorganic form (WHO, 1991). Therefore, a health protective assumption is that the Hg in crops is all in the inorganic form.

Another possible factor to consider is the uptake of methyl mercury (MeHg) by plants. Although it is not expected that Hot Spots facilities would emit MeHg, a fraction of total Hg emitted and deposited to soil could be converted to MeHg in soil. Generally, this may not be a concern in cropland soils, as the content of MeHg would be very low. Nevertheless, results by Gnamus et al. (2001) observed MeHg to be approximately 10 times more phytoavailable then total Hg in an ecotoxicology field study of an Hg-polluted region. Phytoavailability of both total Hg and MeHg increases with decreasing soil pH below 7 and decreased soil content of organic matter.

In rice paddies exposed to Hg smelting and mining facilities, it was found that the percent of total Hg in soil that was MeHg ranged from 0.092 to 0.003 percent (Horvat et al., 2003). However, the percent of total Hg that was MeHg in brown rice grown in the contaminated region ranged from 5 to 84 percent, indicating preferential uptake of MeHg from soil. The resulting UFs for rice ranged from 550 to 6000, suggesting rice may be a high accumulator of MeHg. However, the risk assessment conducted by Horvat et al. (2003) could not establish a clear correlation between total Hg and MeHg in soil and in rice, indicating that uptake and retention of Hg in rice is influenced by a number of factors other than total Hg in soil. Although background mean levels of Hg in U.S. agricultural soils could not be located, a review by Wiersma et al. (1986) showed mean levels of Hg in European and Canadian agricultural soils to be in the range of 0.06 to 0.2 mg/kg dry soil. On average, the concentration of Hg in polluted soils reviewed in this document was about 3.6 mg/kg. Typical dry weight plant concentrations of Hg are listed as 0.001 to 0.3 mg/kg (Vecera et al., 1999). In this

document, the overall Hg concentration in crops grown in Hg-polluted soils was about 0.2 mg/kg.

Table H.6: Percentile distribution for mercury fresh weight soil-to-plant uptake factors

	Leafy	Exposed	Protected	Root
n	33	23	15	18
minimum	0.00021	0.000248	0.000106	0.00111
maximum	0.0813	0.0938	0.0363	0.0588
mean	0.0163	0.00855	0.00804	0.0119
median	0.00875	0.00225	0.00514	0.00553
90th percentile	0.0478	0.0175	0.016	0.0274
95th percentile	0.06	0.0198	0.0223	0.0545

H.8 Nickel

Nickel (Ni) is considered to be one of the more mobile heavy metals in soils (Sauerbeck and Hein, 1991). However, in contrast to Cd, the toxicity of Ni in mammals is lower and phytotoxicity occurs at lower concentrations. Similar to other divalent, cationic metals, acidification of soil increases bioavailability, and liming of soil decreases bioavailability, of Ni to plants. The UF data presented in Table H.7 are based on field-contaminated studies. One study that added Ni salts to soil can be found in the database, but appeared to result in increased plant uptake compared to the field data and was, thus, not included for the UF calculations.

The mean concentration of Ni in uncontaminated U.S. agricultural soils is 23.9 mg/kg, with 5th and 95th percentiles of 4.1 and 56.8 mg/kg, respectively (Holmgren et al., 1993). The mean concentration of Ni for field-contaminated soils reviewed in this document was about 70 mg/kg d.w., with a range of 13 to 122 mg/kg d.w. Typical Ni levels in plants are expected to be in the range of 0.1 to 5 mg/kg dry weight (Vecera et al., 1999). In this report, the overall mean dry weight concentration of Ni in crops was about 9 mg/kg.

Table H.7: Percentile distribution for nickel fresh weight soil-to-plant uptake factors

	Leafy	Exposed	Protected	Root
n	11	13	9	11
minimum	0.00135	0.00025	0.00875	0.00163
maximum	0.0375	0.00625	0.075	0.0175
mean	0.0145	0.00293	0.0305	0.00638
median	0.00888	0.00224	0.025	0.00463
90th percentile	0.0250	0.00610	0.055	0.0125
95th percentile	0.0313	0.00618	0.065	0.0150

H.9 Selenium

The major inorganic species of selenium (Se) in plant sources is selenate, which is translocated directly from the soil and is less readily bound to soil components than selenite (McLaughlin et al., 1999; Rayman, 2008) .The more reduced forms, selenide and elemental Se, are virtually insoluble and do not contribute directly to plant uptake. Other major Se species in plants are biosynthesized, including selenomethionine, smaller amounts of selenocysteine, and Se-containing proteins. At pH values around 7.0 or greater, oxidation to the more soluble selenate ion is favored. Thus, endemic vegetation in alkaline, seleniferous soil of the western U.S. has evolved that is highly tolerant and can hyperaccumulate Se (McLaughlin et al., 1999).

However, potential Se-accumulators that are food sources for humans are largely limited to Brazil nuts, a tree crop that is not grown in California (Rayman et al., 2008). Crops of the Brassica (e.g., broccoli, cabbage) and Allium (e.g., onions, garlic, leeks, chives) families appear to more readily accumulate Se than other crops, and form the Se detoxification products Se-methyl-selenocysteine and gamma-glutamyl-Se-methyl-selenocysteine. Se-enriched plants have been shown in animals to have potent anti-tumor effects that are attributed to these Se detoxification products.

Though there is no direct evidence in humans, it is generally accepted on the basis of animal studies that inorganic forms of Se are more acutely toxic than organic species, selenite being slightly more toxic than selenate (Rayman et al., 2008). In chronic studies of humans, lower toxicity is seen with organically bound Se, although there are limited data on the toxicity of individual compounds.

Selenomethionine is known to be the main Se species present in the diet of Chinese who developed chronic selenosis from consumption of high-Se-containing maize and rice. Based on these Chinese studies, 1540 and 819 µg/day were established as the LOAEL and NOAEL, respectively, for total daily Se intake (Rayman, 2008). However, the levels found in crops rarely accumulate greater than 25-30 µg/g even in seleniferous areas suggesting other sources of Se are also contributors to chronic Se toxicity.

Although the UF data for Se were limited, an overall mean dry weight crop Se concentration of about 4 mg/kg was calculated from the reviewed studies, with a maximum crop concentration of 19 mg/kg. Kloke et al. (1984) observed a general dry weight UF for Se in plants would be 0.1 to 10. Based on the studies examined in this document, an overall dry weight uptake factor of 0.9 was calculated for crops grown in Se-polluted soils, which was within the range predicted. Field contamination studies were the primary source of the UF distribution data in Table H.8. The Se pollution sources included mainly fly ash, smelters and compost.

Table H.8: Percentile distribution for selenium fresh weight soil-to-plant uptake factors

	Leafy	Exposed	Protected	Root
n	12	10	7	10
minimum	0.006	0.00132	0.00625	0.005
maximum	0.25	0.25	1.25	0.375
mean	0.0587	0.0415	0.256	0.0689
median	0.0328	0.0106	0.07	0.0195
90th percentile	0.12	0.104	0.678	0.15
95th percentile	0.179	0.177	0.964	0.263

H.10 Database

The database that lists all of the studies, values, with references is presented as Table H.9-1 through Table H.15-4 in the following pages.

Abbreviations in these tables:

soil conc bckd: the concentration of the chemical in the control soil samples

soil conc contam: the concentration of the chemical in the soil treated with the chemical

tissue conc bckg: the concentration of the chemical in the control tissue samples of the crop

tissue conc contam: the concentration of the chemical in the tissue of the crop grown in the soil treated with the chemical

contam: the related sample treated with the chemical

wt: weight

dw: dry weight

wet w: wet weight

ww: wet weight

Calculation:

$$\text{Uptake factor (contam) dry wt} = \frac{\text{tissue conc contam dry wt} - \text{tissue conc bckg dry wt}}{\text{soil conc contam} - \text{soil conc bckd}}$$

$$\text{Uptake factor (contam) wet wt plant/dw soil} = \text{Uptake factor (contam) dry wt} \times \text{dry-to-wet wt conversion factor}$$

$$\text{Uptake factor (contam) ww plant/wet w soil} = \frac{\text{Uptake factor (contam) wet wt plant/dw soil}}{\text{dry-to-wet weight fraction for soil (0.8)}}$$

Table H.9-1 Arsenic field studies on leafy crops.

Study Type	soil conc bckd mg/kg	soil conc contam mg/kg	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
Field		377	leaf mustard		20	0.05305	0.08	0.004244	0.005305	Clemente et al. (2005)
25% mine waste - greenhouse	23.3	187	lettuce	5.47	21.5	0.11497	0.045	0.005	0.00625	Cobb et al., (2000)
field-fly ash - pot	8.8	9.5	cabbage	0.2	0.3	0.03	0.08	0.003	0.00375	Furr et al. (1978a)
Field		6.04	Chinese cabbage			0.025	0.08	0.002	0.0025	Huang et al. (2006)
Field		6.04	leaf mustard			0.07125	0.08	0.0057	0.007125	Huang et al. 2006
Field		6.04	lettuce			0.046	0.05	0.0023	0.002875	Huang et al. 2006
Field		6.04	pakchoi			0.04625	0.08	0.0037	0.004625	Huang et al. 2006
Field		6.04	water spinach			0.07375	0.08	0.0059	0.007375	Huang et al. 2006
Field			amaranthus			0.55	0.08	0.044	0.055	Huq and Naidu (2005)
Field			cabbage			0.44	0.08	0.0352	0.044	Huq and Naidu 2005
wood preserve. Factory-field	3.4	17.9	kale	0.078	0.1	0.0056	0.08	0.00045	0.000563	Larsen et al., (1992)
wood preserve. Factory-field	3.4	17.9	lettuce	0.048	0.086	0.0048	0.05	0.00024	0.0003	Larsen et al., 1992
mining, smelting-field		446.64	cabbage		1.48	0.0033	0.08	0.00027	0.000338	Li et al., (2006)
mining, smelting-field		446.64	cabbage		1.21	0.0027	0.08	0.00022	0.000275	Li et al., 2006
mining, smelting-field		446.64	Chinese cabbage		1.85	0.0041	0.08	0.00034	0.000425	Li et al., 2006
mining, smelting-field		446.64	spinach		1.37	0.0031	0.08	0.00025	0.000313	Li et al., 2006
Field		6.01	amaranth		0.67	0.11148	0.08	0.008918	0.011148	Liu et al. (2006)
Field		6.01	cabbage		0.81	0.13478	0.08	0.010782	0.013478	Liu et al. 2006
Field		6.01	celery		0.49	0.08153	0.08	0.006522	0.008153	Liu et al. 2006
Field		6.01	Chinese cabbage		0.45	0.07488	0.08	0.00599	0.007488	Liu et al. 2006
Field		6.01	Chinese chive		0.57	0.09484	0.08	0.007587	0.009484	Liu et al. 2006
Field		5.54	leek		0.62	0.11191	0.08	0.008953	0.011191	Liu et al. 2006
field		6.01	pakchoi		3	0.49917	0.08	0.039933	0.049917	Liu et al. 2006
pot	9.83	745	Radish	0.28	14.4	0.01933	0.08	0.001546	0.001933	Mathe-Gaspar and Anton (2002)

Table H.9-1 Arsenic field studies on leafy crops.

Study Type	soil conc bckd mg/kg	soil conc contam mg/kg	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
pot	9.83	745	Radish	0	48.7	0.06537	0.08	0.00523	0.006537	Mathe-Gaspar and Anton 2002
Env polluted soil - field		118	lettuce		7.2	0.06102	0.049	0.003	0.00375	Mattina et al., (2003)
Env polluted soil - field		125.9	spinach		1.55	0.012	0.093	0.0011	0.001375	Mattina et al., 2003

Average Arsenic uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00666±0.00982

Table H.9-2 Arsenic field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-fly ash - pot	8.8	9.5	tomato	0.03	0.1	0.01	0.059	0.0006	0.00075	Furr et al. 1978
field		6.04	bottle gourd			0.00397	0.126	0.0005	0.000625	Huang et al. 2006
field		6.04	cauliflower			0.00873	0.126	0.0011	0.001375	Huang et al. 2006
field		6.04	celery			0.05873	0.126	0.0074	0.00925	Huang et al. 2006
field		6.04	cowpea			0.00272	0.257	0.0007	0.000875	Huang et al. 2006
field		6.04	eggplant			0.00822	0.073	0.0006	0.00075	Huang et al. 2006
field		6.04	onion			0.0088	0.125	0.0011	0.001375	Huang et al. 2006
field		6.04	towel gourd			0.00397	0.126	0.0005	0.000625	Huang et al. 2006
field			bean			0.27	0.111	0.02997	0.037463	Huq and Naidu 2005
field			cauliflower			0.84	0.126	0.10584	0.1323	Huq and Naidu 2005
field			tomato			0.55	0.059	0.03245	0.040563	Huq and Naidu 2005
mining, smelting-field		446.64	capsicum		0.75	0.0017	0.074	0.00013	0.000163	Li et al., 2006
mining, smelting-field		446.64	cucumber		0.49	0.0011	0.039	0.000043	5.38E-05	Li et al., 2006
mining, smelting-field		446.64	eggplant		0.45	0.001	0.073	0.000074	9.25E-05	Li et al., 2006
field		5.54	broccoli		0.59	0.1065	0.126	0.013419	0.016773	Liu et al. 2006
field		6.48	cucumber		0.53	0.08179	0.039	0.00319	0.003987	Liu et al. 2006
field		6.01	Eggplant		0.98	0.16306	0.073	0.011903	0.014879	Liu et al. 2006
field		6.01	kidney bean		2.98	0.49584	0.111	0.055038	0.068798	Liu et al. 2006
field		6.01	pepper		0.39	0.06489	0.126	0.008176	0.01022	Liu et al. 2006
field		6.01	tomato		0.46	0.07654	0.059	0.004516	0.005645	Liu et al. 2006
air dep, mine waste, poll. Water		459.02	capsicum		1.3		0.074	0.00021	0.000263	Liu et al., (2005)
air dep, mine waste, poll. Water	96.92	459.02	string bean	0.54	1.33	0.0029	0.111	0.00032	0.0004	Liu et al., 2005

Average Arsenic uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0158±0.0313

Table H.9-3 Arsenic field studies on protected crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
25% mine waste - greenhouse	23.3	187	bush bean	0.184	0.304	0.099	0.00016	0.0002	Cobb et al., 2000
field-fly ash - pot	8.8	9.5	corn	0.1	0.2	0.895	0.02	0.025	Furr et al. 1978
field			cowpea			0.257	0.03341	0.041763	Huq and Naidu 2005
field			garlic			0.222	0.12654	0.158175	Huq and Naidu 2005
field			pea			0.257	0.21331	0.266638	Huq and Naidu 2005
field			pumpkin			0.222	0.03108	0.03885	Huq and Naidu 2005
mining, smelting-field		446.64	pumpkin		0.5	0.082	0.000092	0.000115	Li et al., 2006
air dep, mine waste, poll. Water		459.02	corn		0.21	0.261	0.00012	0.00015	Liu et al., 2005

Average Arsenic uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0664±0.0962

Table H.9-4 Arsenic field studies on root crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-ground water		13.3 (4-14)	potato		0.8	0.0602	0.222	0.013364	0.016706	Alam et al. (2003)
25% mine waste - greenhouse	23.3	187	radish	0.593	2.94	0.01572	0.047	0.00075	0.000938	Cobb et al., 2000
field-fly ash - pot	8.8	9.5	carrot (peeled)	0.05	0.2	0.02	0.118	0.002	0.0025	Furr et al. 1978
field-fly ash - pot	8.8	9.5	Onion (peeled)	0.1	0.3	0.03	0.125	0.004	0.005	Furr et al. 1978
field-fly ash - pot	8.8	9.5	Potato (peeled)	0.1	0.1	0.01	0.222	0.002	0.0025	Furr et al. 1978
field		6.04	garlic			0.0245	0.2	0.0049	0.006125	Huang et al. 2006
field		6.04	radish			0.0285	0.2	0.0057	0.007125	Huang et al. 2006
field		6.04	taro			0.0165	0.2	0.0033	0.004125	Huang et al. 2006
field			carrot			0.23	0.118	0.02714	0.033925	Huq and Naidu 2005
field			radish			0.18	0.2	0.036	0.045	Huq and Naidu 2005
wood preserve. Factory-field	3.4	17.9	carrot (unpeeled)	0.032	0.042	0.0023	0.118	0.00027	0.000338	Larsen et al., 1992
wood preserve. Factory-field	3.4	17.9	potato (unpeeled)	0.037	0.077	0.0043	0.222	0.00095	0.001188	Larsen et al., 1992
field		5.54	carrot		0.15	0.02708	0.118	0.003195	0.003994	Liu et al. 2006
field		6.01	radish		0.22	0.03661	0.2	0.007321	0.009151	Liu et al. 2006
landfill-field		27	carrot (unpeeled)		0.17	0.0063	0.106	0.00067	0.000838	Samsoe-Petersen et al., (2002)
landfill-field		27	potato (unpeeled)		0.127	0.0047	0.094	0.00044	0.00055	Samsoe-Petersen et al., 2002
landfill-field		27	radish		0.27	0.01	0.059	0.00059	0.000738	Samsoe-Petersen et al., 2002

Average Arsenic uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00828±0.0129

Table H.10-1 Cadmium field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field	0.69	1.6	amaranth	0.81	3.85	2.406	0.08	0.1925	0.2406	Hu and Ding (2009)
field		0.16	amaranth		0.16	1.000	0.08	0.0800	0.1000	Liu et al. 2006
indust. Poll. Depo. - field		12	amaranthus		5.66	0.470	0.08	0.0380	0.0475	Pandey and Pandey, (2009)
Indust. sewage wastes - field	0.5	22	amaranthus	0.14	1.1	0.050	0.08	0.0040	0.0050	Srikanth et al., (1991)
field-wastewater	0.12	0.87	basil	0.16	0.6	0.690	0.08	0.0550	0.0688	Shariatpanahi and Anderson (1986)
field		4.4	cabbage		0.3	0.068	0.08	0.0055	0.0068	Chumbley and Unwin (1982)
sewage sludge - pots		23.22	cabbage		1.77	0.076	0.08	0.0061	0.0076	Jackson & Alloway, (1991)
mining, smelting-field		7.43	cabbage		0.71	0.096	0.08	0.0077	0.0096	Li et al., 2006
mining, smelting-field		7.43	cabbage		1.29	0.170	0.08	0.0130	0.0163	Li et al., 2006
field		0.16	cabbage		0.076	0.475	0.08	0.0380	0.0475	Liu et al. 2006
sewage sludge - field		10.5	cabbage		2.1	0.200	0.08	0.0200	0.0250	Muntau et al., (1987)
Indust. sewage wastes - field	0.5	22	cabbage	0.02	2.88	0.130	0.078	0.0100	0.0125	Srikanth et al., 1991
field - smelter	0.108	4.99	cabbage				0.052	0.1740	0.2175	Zheng et al. (2007a)a
field		1.6	celery		3.57	2.231	0.08	0.1785	0.2231	Hu and Ding 2009
field		0.16	celery		0.1	0.625	0.08	0.0500	0.0625	Liu et al. 2006
field - smelter	0.108	12.5	celery				0.058	0.1310	0.16375	Zheng et al. 2007a
mining, smelting-field		7.43	Chinese cabbage		1.31	0.180	0.08	0.0130	0.0163	Li et al., 2006
field		0.16	Chinese cabbage		0.2	1.250	0.08	0.1000	0.1250	Liu et al. 2006
field		0.515	Chinese cabbage		0.2625	0.510	0.08	0.0408	0.0510	Wang et al. (2006)
field - smelter	0.108	22.8	Chinese cabbage				0.055	0.1280	0.16	Zheng et al. 2007a
field		0.16	Chinese chive		0.12	0.750	0.08	0.0600	0.0750	Liu et al. 2006
sewage sludge-field-grnhs		2.55	chinese leek		0.9	0.350	0.089	0.0310	0.0388	Yang et al., (2009)
field-wastewater	0.12	0.87	garden cress	0.1	0.6	0.690	0.08	0.0550	0.0688	Shariatpanahi and Anderson 1986
field - smelter	0.108	43.4	green				0.085	0.0440	0.055	Zheng et al. 2007a

Table H.10-1 Cadmium field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
			onion							
field		0.17	leek		0.055	0.324	0.08	0.0259	0.0324	Liu et al. 2006
field - smelter	0.108	39.2	leek			2.250	0.08	0.1800	0.2250	Zheng et al. 2007a
field		7.8	lettuce		4.2	0.538	0.05	0.0269	0.0337	Chumbley and Unwin 1982
25% mine waste - greenhouse	1.38	6.06	lettuce	1.61	5.37	0.890	0.045	0.0400	0.0500	Cobb et al., 2000
Env. contam. Soil 1a - potted		1.8	lettuce		2.5	1.400	0.049	0.0686	0.0858	Crews & Davies, (1985)
Env. contam. Soil 1b - potted		2.2	lettuce		7.8	3.500	0.049	0.1715	0.2144	Crews & Davies, 1985
Env. contam. Soil 2 - potted		4.5	lettuce		11.8	2.600	0.049	0.1274	0.1593	Crews & Davies, 1985
Env. contam. Soil 3 - potted		5.5	lettuce		20.5	3.700	0.049	0.1813	0.2266	Crews & Davies, 1985
field	0.69	1.6	lettuce	1.49	4.19	2.619	0.05	0.1309	0.1637	Hu and Ding 2009
fertilizer	0.53	0.6-0.86	lettuce				0.05	0.1950	0.2438	Huang et al. (2003)
fertilizer in field			lettuce				0.05	0.3199	0.3998	Huang et al. (2004)
sewage sludge - pots		23.22	lettuce		10.57	0.460	0.05	0.0230	0.0288	Jackson & Alloway, 1991
Env polluted soil - field		1	lettuce		2.6	2.600	0.049	0.1274	0.1593	Mattina et al., 2003
sewage sludge-field		2.2	lettuce		2.8	1.300	0.05	0.0650	0.0813	Preer et al., (1995)
smelter area - urban gardens	0.8	12.6	lettuce	0.41	7.55	0.600	0.049	0.0294	0.0368	Pruvot et al., (2006)
landfill-field		2.4	lettuce		0.552	0.230	0.05	0.0115	0.0144	Samsoe-Petersen et al., 2002
moderate urban poll -field		0.56	lettuce		0.21	0.400	0.05	0.0200	0.0250	Samsoe-Petersen et al., 2002
fertilizer-field	ND	0.311	lettuce	ND	0.06	0.200	0.05	0.0100	0.0125	(Schroeder and Balassa, 1963)
fertilizer-field	ND	0.311	lettuce	ND	0.5	1.600	0.045	0.0720	0.0900	Schroeder & Balassa, 1963
urban gardens-field-to-grnhs	0.08	3.28	lettuce	0.65	1.73	0.760	0.045	0.0342	0.0428	Sterrett et al., (1996)
field - smelter	0.108	4.99	lettuce				0.042	0.2030	0.25375	Zheng et al. 2007
field-wastewater	0.12	0.87	mint	0.11	0.7	0.800	0.08	0.0640	0.0800	Shariatpanahi and Anderson 1986
field - smelter	0.108	20.1	mustard				0.071	0.0870	0.10875	Zheng et al. 2007
field		1.6	pakchoi		2.53	1.581	0.08	0.1265	0.1581	Hu and Ding 2009
field		0.16	pakchoi		0.11	0.688	0.08	0.0550	0.0688	Liu et al. 2006
field		0.515	Pakchoi		0.275	0.534	0.08	0.0427	0.0534	Wang et al. 2006
field		15.8	Pakchoi		0.21	0.090	0.08	0.0072	0.0090	Yan et al. (2007)

Table H.10-1 Cadmium field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
sewage sludge-field-greenhouse		2.55	pakchoi		1.25	0.490	0.076	0.0370	0.0463	Yang et al., 2009
field (industrial sewage irrigation)		2.69	palak (spinach)		1.5	0.560	0.08	0.0450	0.0563	Kumar Sharma et al., 2007
field (industrial sewage irrigation)		2.26	palak (spinach)		2.1	0.930	0.08	0.0740	0.0925	Kumar Sharma et al., 2007
field (industrial sewage irrigation)		2.8	palak (spinach)		2.85	1.000	0.08	0.0800	0.1000	Kumar Sharma et al., 2007
pot	0.167	30.5	Radish	0.388	8.78	0.288	0.08	0.0230	0.0288	Mathe-Gaspar and Anton 2002
pot	0.167	30.5	Radish	0.448	9.05	0.297	0.08	0.0237	0.0297	Mathe-Gaspar and Anton 2002
flooded gardens		1.31	sorrel		0.115	0.088	0.08	0.0070	0.0088	Sipter et al. (2008)
non-flooded gardens		0.43	sorrel		0.101	0.235	0.08	0.0188	0.0235	Sipter et al. 2008
field		4.6	spinach		4.6	1.000	0.08	0.0800	0.1000	Chumbley and Unwin 1982
high-Cd fertilizer - greenhouse	0.25	0.2625	spinach	1.48	2.18	8.300	0.08	0.6600	0.8250	He and Singh (1994)
high-Cd fertilizer - greenhouse	0.25	0.2625	spinach	2.32	2.85	10.860	0.08	0.8700	1.0875	He and Singh 1994
low-Cd fertilizer - greenhouse	0.25	0.2527	spinach	1.48	1.74	6.890	0.08	0.5500	0.6875	He and Singh 1994
low-Cd fertilizer - greenhouse	0.25	0.2527	spinach	2.32	2.58	10.210	0.08	0.8200	1.0250	He and Singh 1994
sewage sludge-field	0.48	5.32	spinach	0.94	12.76	1.991	0.08	0.1600	0.2000	Hooda et al., 1997
sewage sludge-field	1.6	4.3	spinach	0.01	0.14	0.030	0.08	0.0030	0.0038	Jamali et al., 2007
mining, smelting-field		7.43	spinach		1.06	0.140	0.08	0.0110	0.0138	Li et al., 2006
field (sewage-fed lake irrigation)			Spinach			2.500	0.08	0.2000	0.2500	Lokeshwari and Chandrappa 2006
Env polluted soil - field		0.7	spinach		5.3	7.600	0.093	0.7000	0.8750	Mattina et al., 2003
indust. Poll. Depo. - field		12	spinach		5.84	0.490	0.08	0.0390	0.0488	Pandey and Pandey, 2009
Indust. sewage wastes - field	0.5	22	spinach	0.13	6.4	0.290	0.086	0.0250	0.0313	Srikanth et al., 1991
field - smelter	0.108	43.4	spinach				0.088	0.0980	0.1225	Zheng et al. 2007
field		9.3	spring greens		1.1	0.118	0.08	0.0095	0.0118	Chumbley and Unwin 1982

Table H.10-1 Cadmium field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
sewage sludge - chamber	0.9	8.4	Swiss chard	2.2	11.2	1.300	0.08	0.1000	0.1250	Mahler et al., 1987
sewage sludge + limed - chamber	0.9	8.4	Swiss chard	1.7	8.4	1.000	0.08	0.0800	0.1000	Mahler et al., 1987
fertilizer-field greenhouse	0.07	1.13	Swiss chard	0.26	1.61	1.400	0.08	0.1000	0.1250	Mulla et al., (1980)
drilling fluid-greenhouse	0.6	19.4	swiss chard	1.5	26.9	1.400	0.08	0.1000	0.1250	Nelson et al., (1984)
sewage sludge-field		2.2	Swiss chard		3.15	1.400	0.08	0.1000	0.1250	Preer et al., 1995
field-wastewater	0.12	0.87	tarragon	0.14	0.05	0.060	0.08	0.0046	0.0058	Shariatpanahi and Anderson 1986
field		0.515	Water spinach		0.3625	0.704	0.08	0.0563	0.0704	Wang et al. 2006
field survey						0.507	0.08	0.0406	0.0507	Cambra et al. 1999

Average cadmium uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.139±0.214

Table H.10-2 Cadmium field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt mg/kg	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field - smelter	0.108	39.2	aubergine			0.513	0.081	0.0416	0.0519	Zheng et al. 2007a
indust. sewage-field-Egypt	ND	28	bell pepper		0.05	0.002	0.074	0.0001	0.0001	Gorbunov et al., 2003
field - smelter	0.108	20.1	bitter melon				0.066	0.0050	0.00625	Zheng et al. 2007a
landfill-field		2	blackberry					0.0025	0.0031	Samsoe-Petersen et al., 2002
field		0.17	broccoli		0.048	0.282	0.126	0.0356	0.0445	Liu et al. 2006
mining, smelting-field		7.43	capsicum		0.41	0.055	0.074	0.0040	0.0050	Li et al., 2006
air dep, mine waste, poll. Water		6.77	capsicum		1.37	0.200	0.074	0.0150	0.0188	Liu et al., 2005
field - smelter	0.108	39.2	capsicum			0.258	0.066	0.0170	0.0213	Zheng et al. 2007a
field		3.5	cauliflower		0.7	0.200	0.126	0.0252	0.0315	Chumbley and Unwin 1982
indust. sewage-field-Egypt	ND	28	cucumber		0.06	0.002	0.039	0.0001	0.0001	Gorbunov et al., 2003
mining, smelting-field		7.43	cucumber		0.66	0.089	0.039	0.0035	0.0044	Li et al., 2006
field		0.16	cucumber		0.059	0.369	0.039	0.0144	0.0180	Liu et al. 2006
sewage sludge-field-grnhs		2.55	cucumber		0.2	0.080	0.04	0.0031	0.0039	Yang et al., 2009
mining, smelting-field		7.43	eggplant		0.4	0.054	0.073	0.0039	0.0049	Li et al., 2006
field		0.16	Eggplant		0.16	1.000	0.073	0.0730	0.0913	Liu et al. 2006
indust. Poll. Depo. - field		12	eggplant		4.18	0.350	0.073	0.0260	0.0325	Pandey and Pandey, 2009
field		0.515	Eggplant		0.3	0.638	0.073	0.0466	0.0583	Wang et al. 2006
indust. sewage-field-Egypt	ND	28	fig		0.015	0.001	0.126	0.0001	0.0001	Gorbunov et al., 2003
sewage sludge-field	1.6	4.3	Indian squash	0.08	0.24	0.060	0.082	0.0050	0.0063	Jamali et al., (2007)
field		0.16	kidney bean		0.036	0.225	0.111	0.0250	0.0312	Liu et al. 2006
field-wastewater	0.12	0.87	leek	0.14	0.5	0.570	0.12	0.0690	0.0863	Shariatpanahi and Anderson 1986
indust. sewage-field-Egypt	ND	28	olive		0.03	0.001	0.126	0.0001	0.0001	Gorbunov et al., 2003
landfill-field		2	pear					0.0034	0.0043	Samsoe-Petersen et al., 2002
sewage sludge-field			pepper				0.0408	0.0290	0.0362	Giordano et al., (1979)
field		0.16	pepper		0.15	0.938	0.126	0.1181	0.1477	Liu et al. 2006
field survey			peppers			0.053	0.126	0.0066	0.0083	Cambra et al. (1999)
landfill-field		2	plum					0.0006	0.0008	Samsoe-Petersen et al., 2002
sewage sludge-field			squash				0.082	0.0098	0.0123	Giordano et al., 1979

Table H.10-2 Cadmium field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt mg/kg	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
flooded gardens		1.31	squash		0.033	0.025	0.082	0.0021	0.0026	Sipter et al. 2008
non-flooded gardens		0.43	squash		0.005	0.012	0.082	0.0010	0.0012	Sipter et al. 2008
air dep, mine waste, poll. Water	2.08	6.77	string bean	0.21	0.67	0.099	0.111	0.0110	0.0138	Liu et al., 2005
25% mine waste - greenhouse	1.38	6.06	tomato	0.523	0.704	0.120	0.065	0.0078	0.0098	Cobb et al., 2000
field		0.15	tomato		0.11	0.733	0.059	0.0433	0.0541	Liu et al. 2006
indust. Poll. Depo. - field		12	tomato		4.96	0.410	0.059	0.0240	0.0300	Pandey and Pandey, 2009
smelter area - urban gardens	0.8	12.6	tomato	0.15	1.23	0.098	0.065	0.0063	0.0079	Pruvot et al., 2006
flooded gardens		1.31	tomato		0.06	0.046	0.059	0.0027	0.0034	Sipter et al. 2008
non-flooded gardens		0.43	tomato		0.008	0.019	0.059	0.0011	0.0014	Sipter et al. 2008
smelter contam - field	0.08	4.4	tomato		0.43	0.098	0.065	0.0064	0.0080	Tomov & Alandjiyski, (2006)
sewage sludge-field-grnhs		2.55	tomato		0.2	0.080	0.033	0.0026	0.0033	Yang et al., 2009
field - smelter	0.11	43.4	tomato				0.056	0.0030	0.00375	Zheng et al. 2007a
field		0.515	Towel gourd		0.0976	0.189	0.082	0.0155	0.0194	Wang et al. 2006

Average cadmium uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0216±0.0304

Table H.10-3 Cadmium field studies on protected crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant /wet w soil	References
flooded gardens		1.31	bean		0.02	0.01527	0.111	0.0016947	0.0021	Sipter et al. 2008
non-flooded gardens		0.43	bean		0.01	0.02326	0.111	0.0025814	0.0032	Sipter et al. 2008
indust. sewage-field-Egypt	ND	28	bean (spot)		0.28	0.01	0.111	0.001	0.0013	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	28	bean (white)		0.26	0.009	0.111	0.001	0.0013	Gorbunov et al., 2003
sewage sludge-pot-field		4.6	beans		0.27	0.06	0.222	0.013	0.0163	Sauerbeck, 1991
field survey			broad beans			0.0108	0.126	0.0013608	0.0017	Cambra et al. 1999
25% mine waste - grhs	1.38	6.06	bush bean	0.145	0.01	0.0017	0.099	0.00017	0.0002	Cobb et al., 2000
sewage sludge-field			cantelope				0.06	0.0192	0.0240	Giordano et al., 1979
sewage sludge-field	1.6	4.3	cluster beans	0.04	0.2	0.05	0.111	0.005	0.0063	Jamali et al., 2007
field	0.26	25.3889	corn		0.2	0.00788	0.261	0.002056	0.0026	Bi et al. (2006)
air dep, mine waste, poll. Water		6.77	corn		0.47	0.069	0.261	0.018	0.0225	Liu et al., 2005
indust. sewage-field	0.072	3.72	corn	0.002	0.23	0.062	0.895	0.055	0.0688	Nan et al., (2002)
smelter area - ag field	0.4	8.1	corn	0.07	0.18	0.022	0.273	0.0062	0.0078	Pruvot et al., 2006
field		0.515	Cowpea		0.02724	0.05289	0.257	0.0135922	0.0170	Wang et al. 2006
field - smelter	0.108	43.4	cowpea				0.097	0.004	0.005	Zheng et al. 2007a
landfill-field		2	green bean		0.098	0.041	0.027	0.0011	0.0014	Samsoe-Petersen et al., 2002
moderate urban poll -field		0.56	green bean		0.009	0.02	0.111	0.002	0.0025	Samsoe-Petersen et al., 2002
landfill-field		2	hazelnut					0.004	0.0050	Samsoe-Petersen et al., 2002
field - smelter	0.108	39.2	kidney bean			0.119	0.103	0.012257	0.0153	Zheng et al. 2007a
fertilizer-field	ND	0.311	onion	ND	0.024	0.08	0.125	0.01	0.0125	Schroeder & Balassa, 1963
fertilizer-field	ND	0.311	pea	ND	0.04	0.1	0.257	0.03	0.0375	Schroeder & Balassa, 1963
sewage sludge-field	1.6	4.3	peas	0.075	0.2	0.05	0.257	0.01	0.0125	Jamali et al., 2007
sewage sludge-pot-field		4.6	peas		0.2	0.04	0.257	0.01	0.0125	Sauerbeck, 1991
mining, smelting-field		7.43	pumpkin		0.46	0.062	0.082	0.0051	0.0064	Li et al., 2006
field - smelter	0.108	43.4	pumpkin				0.065	0.001	0.001	Zheng et al. 2007a
fertilizer-field	ND	0.311	string bean	ND	0.015	0.05	0.111	0.01	0.0125	Schroeder & Balassa, 1963
field		7.8	sweet corn		1.5	0.19231	0.261	0.0501923	0.0627	Chumbley and Unwin 1982

Average cadmium uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0134±0.0175

Table H.10-4 Cadmium field studies on root crops.

Study Type	soil conc bcgd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bcgd(T) dry wt (mg/kg)	tissue conc contam(C) dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
fertilizer-field	ND	0.311	beet	ND	0.045	0.100	0.2	0.0300	0.0375	Schroeder & Balassa, 1963
field		6.5	beetroot		2	0.308	0.222	0.0683	0.0854	Chumbley and Unwin 1982
smelter - field - home gardens		40.6	carrot		4.4	0.110	0.118	0.0130	0.0163	Chaney et al., (1988)
sewage sludge-field	0.48	5.32	carrot	0.63	1.71	0.350	0.118	0.0410	0.0513	Hooda et al., 1997
field		0.17	carrot		0.085	0.500	0.118	0.0590	0.0738	Liu et al. 2006
indust. Poll. Depo. - field		12	carrot		2.06	0.170	0.118	0.0200	0.0250	Pandey and Pandey, 2009
smelter area - urban gardens	0.8	12.6	carrot	0.085	1.53	0.120	0.118	0.0140	0.0175	Pruvot et al., 2006
fertilizer-field	ND	0.311	carrot	ND	0.068	0.200	0.118	0.0300	0.0375	Schroeder & Balassa, 1963
flooded gardens		1.31	carrot		0.13	0.099	0.118	0.0117	0.0146	Sipter et al. 2008
non-flooded gardens		0.43	carrot		0.068	0.158	0.118	0.0187	0.0233	Sipter et al. 2008
contam-irrig. water - greenhouse		3.6	carrot		1.22	0.340	0.135	0.0460	0.0575	Zheng et al., (2008)
sewage sludge-field-greenhouse		2.55	carrot		0.7	0.270	0.11	0.0300	0.0375	Yang et al., 2009
field - smelter	0.108	39.2	carrot			0.752	0.088	0.0662	0.0827	Zheng et al. 2007a
high-Cd fertilizer - greenhouse	0.25	0.2625	carrot	0.115	0.145	0.550	0.118	0.0650	0.0813	He and Singh 1994
high-Cd fertilizer - greenhouse	0.25	0.2625	carrot	0.125	0.165	0.630	0.118	0.0740	0.0925	He and Singh 1994
low-Cd fertilizer - greenhouse	0.25	0.2527	carrot	0.115	0.135	0.530	0.118	0.0630	0.0788	He and Singh 1994
low-Cd fertilizer - greenhouse	0.25	0.2527	carrot	0.125	0.15	0.590	0.118	0.0700	0.0875	He and Singh 1994
fertilizers w/ Cd		0.3	carrot (unpeeled)		0.25	0.800	0.11	0.0900	0.1125	Jansson and Oborn, (2000)
landfill-field		2.4	carrot (unpeeled)		0.26	0.110	0.127	0.0140	0.0175	Samsoe-Petersen et al., 2002
moderate urban poll -field		0.56	carrot (unpeeled)		0.12	0.200	0.118	0.0300	0.0375	Samsoe-Petersen et al., 2002
sewage sludge-pot-field		4.6	carrots		0.9	0.200	0.118	0.0200	0.0250	Sauerbeck, 1991
field survey			chard			0.519	0.2	0.1038	0.1298	Cambra et al. 1999
indust. sewage-field-Egypt	ND	28	garlic		0.21	0.008	0.125	0.0009	0.0011	Gorbunov et al., 2003
smelter area - urban gardens	0.8	12.6	leek	0.14	1.58	0.130	0.146	0.0180	0.0225	Pruvot et al., 2006
field		3.1	leeks		0.8	0.258	0.2	0.0516	0.0645	Chumbley and Unwin 1982
indust. sewage-field-Egypt	ND	28	onion		0.27	0.010	0.125	0.0010	0.0013	Gorbunov et al., 2003
field-wastewater	0.12	0.87	onion	0.12	0.3	0.340	0.125	0.0400	0.0500	Shariatpanahi and Anderson 1986
flooded gardens		1.31	onion		0.07	0.053	0.125	0.0067	0.0083	Sipter et al. 2008
non-flooded gardens		0.43	onion		0.056	0.130	0.125	0.0163	0.0203	Sipter et al. 2008
field survey			onions			0.105	0.125	0.0132	0.0164	Cambra et al. 1999

Table H.10-4 Cadmium field studies on root crops.

Study Type	soil conc bcgd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bcgd(T) dry wt (mg/kg)	tissue conc contam(C) dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
fertilizer-field	ND	0.311	parsnip	0.15	0.7	2.200	0.2	0.5000	0.6250	Schroeder & Balassa, 1963
smelter - field - home gardens		13.2	potato		3.6	0.270	0.202	0.7300	0.9125	Chaney et al., 1988
field		10.8	potato		0.6	0.056	0.222	0.0123	0.0154	Chumbley and Unwin 1982
smelter flue-dust	0.3	106.5	potato	0.16	1.67	0.016	0.222	0.0035	0.0044	Dudka et al. 1996
smelter flue-dust	0.3	54.4	potato	0.16	2.12	0.039	0.222	0.0087	0.0108	Dudka et al. 1996
smelter flue-dust	0.3	7.1	potato	0.16	0.53	0.075	0.222	0.0166	0.0207	Dudka et al. 1996
smelter flue-dust	0.3	3.2	potato	0.16	0.42	0.131	0.222	0.0291	0.0364	Dudka et al. 1996
smelter area - ag field	0.4	8.1	potato	0.3	0.45	0.056	0.202	0.0110	0.0138	Pruvot et al., 2006
smelter area - urban gardens	0.8	12.6	potato	0.05	0.54	0.043	0.202	0.0087	0.0109	Pruvot et al., 2006
fertilizer-field	ND	0.311	potato	ND	0.015	0.050	0.222	0.0100	0.0125	Schroeder & Balassa, 1963
smelter contam - field	0.08	4.4	potato		0.097	0.022	0.202	0.0044	0.0055	Tomov & Alandjiyski, 2006
sewage sludge - pots		23.22	potato (peeled)		0.3	0.013	0.222	0.0029	0.0036	Jackson & Alloway, 1991
sewage sludge-field		2.77	potato (peeled)		0.07	0.030	0.218	0.0055	0.0069	Smith (1994)
landfill-field		2.4	potato (unpeeled)		0.089	0.037	0.135	0.0050	0.0063	Samsoe-Petersen et al., 2002
moderate urban poll -field		0.56	potato(unpeeled)		0.05	0.090	0.222	0.0200	0.0250	Samsoe-Petersen et al., 2002
field		2.7	radish		1.7	0.630	0.222	0.1398	0.1747	Chumbley and Unwin 1982
25% mine waste - greenhouse	1.38	6.06	radish	0.01	2.31	0.380	0.047	0.0180	0.0225	Cobb et al., 2000
indust. sewage-field-Egypt	ND	28	radish		0.28	0.010	0.085	0.0009	0.0011	Gorbunov et al., 2003
field		0.16	radish		0.083	0.519	0.2	0.1038	0.1297	Liu et al. 2006
field (sewage-fed lake irrigation)			Radish			1.600	0.2	0.3200	0.4000	Lokeshwari and Chandrappa 2006
indust. Poll. Depo. - field		12	radish		2.61	0.220	0.085	0.0190	0.0238	Pandey and Pandey, 2009
smelter area - urban gardens	0.8	12.6	radish	0	2.12	0.170	0.047	0.0079	0.0099	Pruvot et al., 2006
landfill-field		2.4	radish		0.19	0.080	0.041	0.0033	0.0041	Samsoe-Petersen et al., 2002
moderate urban poll -field		0.56	radish		0.071	0.100	0.085	0.0100	0.0125	Samsoe-Petersen et al., 2002
sewage sludge-pot-field		4.6	radish		1.1	0.200	0.05	0.0100	0.0125	Sauerbeck, 1991
fertilizer-field	ND	0.311	radish	ND	0.1	0.300	0.2	0.0600	0.0750	Schroeder & Balassa, 1963
field-wastewater	0.12	0.87	radish	0.18	0.45	0.520	0.085	0.0400	0.0500	Shariatpanahi and Anderson 1986
contam-irrig. water - greenhouse		3.6	radish		1.09	0.300	0.083	0.0250	0.0313	Zheng et al., 2008
sewage sludge-field-greenhouse		2.55	radish		0.5	0.200	0.05	0.0098	0.0123	Yang et al., 2009

Table H.10-4 Cadmium field studies on root crops.

Study Type	soil conc bcgd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bcgd(T) dry wt (mg/kg)	tissue conc contam(C) dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field		4.8	salad onions		1	0.208	0.125	0.0260	0.0326	Chumbley and Unwin 1982
fertilizer-field	ND	0.311	turnip	ND	0.15	0.500	0.2	0.1000	0.1250	Schroeder & Balassa, 1963
field - smelter	0.108	39.2	turnip			0.027	0.108	0.0029	0.0036	Zheng et al. 2007a

Average cadmium uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0683±0.144

Table H.11-1 Lead field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt mg/kg	tissue conc contam dry wt mg/kg	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
pots -env. chamber	30	300	cabbage		2.4	0.0080	0.08	0.0006	0.00075	Caille et al., 2005
pots -env. chamber	30	300	rape		2.3	0.0080	0.08	0.0006	0.00075	Caille et al., 2005
field		117	cabbage		0.3	0.0026	0.08	0.000205	0.0002564	Chumbley and Unwin 1982
field		155	lettuce		2.3	0.0148	0.05	0.000742	0.0009274	Chumbley and Unwin 1982
field		124	spinach		3.7	0.0298	0.08	0.002387	0.0029839	Chumbley and Unwin 1982
field		214	spring greens		2.3	0.0107	0.08	0.00086	0.0010748	Chumbley and Unwin 1982
field		532	leaf mustard		21	0.0395	0.08	0.003158	0.0039474	Clemente et al. 2005
25% mine waste - grnhs	60.9	3600	lettuce	29.8	227	0.0631	0.045	0.002838	0.0035469	Cobb et al., 2000
Env. contam. Soil 1a - potted - outside		301	lettuce		2	0.0066	0.049	0.000326	0.000407	Crews & Davies, 1985
Env. contam. Soil 1b - potted - outside		169	lettuce		7.7	0.0456	0.049	0.002233	0.0027907	Crews & Davies, 1985
Env. contam. Soil 2 - potted - outside		754	lettuce		5.7	0.0076	0.049	0.00037	0.000463	Crews & Davies, 1985
Env. contam. Soil 3 - potted - outside		850	lettuce		14.3	0.0168	0.049	0.000824	0.0010304	Crews & Davies, 1985
urban gardens-field			cilantro				0.08	0.002	0.0025	Finster et al., 2004
urban gardens-field			collard greens				0.147	0.0004	0.0005	Finster et al., 2004
urban gardens-field			coriander				0.08	0.003	0.00375	Finster et al., 2004
urban gardens-field			ipasote				0.08	0.002	0.0025	Finster et al., 2004
urban gardens-field			lemon balm				0.08	0.001	0.00125	Finster et al., 2004
urban gardens-field			mint				0.08	0.0009	0.001125	Finster et al., 2004
urban gardens-field			rhubarb				0.052	0.00047	0.0005875	Finster et al., 2004
urban gardens-field			Swiss chard				0.089	0.0027	0.003375	Finster et al., 2004
sewage sludge-field	70	259	spinach	0.82	0.95	0.0080	0.08	0.0006	0.00075	Hooda et al., 1997
field	65.9	361	amaranth	2.66	45.7	0.1266	0.08	0.010127	0.0126593	Hu and Ding 2009
field		361	celery		22.1	0.0612	0.08	0.004898	0.0061219	Hu and Ding 2009
field	65.9	361	lettuce	1.14	37.5	0.1039	0.05	0.005194	0.0064924	Hu and Ding 2009
field		361	pakchoi		36.2	0.1003	0.08	0.008022	0.0100277	Hu and Ding 2009
Pb arsenate - grnhs	60.9	342.3	lettuce	10.2	12.5	0.0400	0.05	0.002	0.0025	Hutchinson et al. 1974

Table H.11-1 Lead field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt mg/kg	tissue conc contam dry wt mg/kg	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
sewage sludge-field	21.1	67.4	spinach	0.33	1.2	0.0200	0.08	0.001	0.00125	Jamali et al., 2007
mining, smelting-field		223.22	cabbage			0.0500	0.08	0.004	0.005	Li et al., 2006
mining, smelting-field		223.22	cabbage			0.0490	0.08	0.0039	0.004875	Li et al., 2006
mining, smelting-field		223.22	Chinese cabbage			0.0780	0.08	0.0062	0.00775	Li et al., 2006
mining, smelting-field		223.22	spinach			0.0700	0.08	0.0056	0.007	Li et al., 2006
field		14.48	amaranth		1.91	0.1319	0.08	0.010552	0.0131906	Liu et al. 2006
field		14.48	cabbage		1	0.0691	0.08	0.005525	0.0069061	Liu et al. 2006
field		14.48	celery		1.76	0.1215	0.08	0.009724	0.0121547	Liu et al. 2006
field		14.48	Chinese cabbage		2.05	0.1416	0.08	0.011326	0.0141575	Liu et al. 2006
field		14.48	Chinese chive		2.53	0.1747	0.08	0.013978	0.0174724	Liu et al. 2006
field		14.48	pakchoi		2.02	0.1395	0.08	0.01116	0.0139503	Liu et al. 2006
pot	18.5	2897	Radish	2.9	94.3	0.0326	0.047	0.00153	0.0019124	Mathe-Gaspar and Anton 2002
pot	18.5	2897	Radish	2.4	272.4	0.0940	0.047	0.004419	0.0055242	Mathe-Gaspar and Anton 2002
sewage sludge - field		775	cabbage		0.31	0.0004	0.08	0.00003	0.0000375	Muntau et al., 1987
drilling fluid-grnhs	17	1131	swiss chard	1.7	9.2	0.0080	0.08	0.0007	0.000875	Nelson et al., 1984
Env. contam. Soil (paint?) - potted - grnhs		2000	collard		8	0.0040	0.147	0.0006	0.00075	Nicklow et al., (1983)
Env. contam. Soil (paint?) - potted - grnhs		2000	kale		7	0.0035	0.173	0.0006	0.00075	Nicklow et al., 1983
Env. contam. Soil (paint?) - potted - grnhs		2000	lettuce		25	0.0125	0.049	0.000613	0.0007656	Nicklow et al., 1983
indust. Poll. Depo. - field		165.85	amaranthus		18.44	0.1100	0.08	0.0088	0.011	Pandey and Pandey, 2009
indust. Poll. Depo. - field		165.85	spinach		19.58	0.1200	0.08	0.0096	0.012	Pandey and Pandey, 2009
sewage sludge-field		98	lettuce			0.0200	0.05	0.001	0.00125	Preer et al., 1995
sewage sludge-field		98	Swiss chard			0.0300	0.08	0.003	0.00375	Preer et al., 1995
smelter area - urban gardens - field	84	872	lettuce	2.24	6.93	0.0079	0.049	0.000387	0.0004839	Pruvot et al., 2006

Table H.11-1 Lead field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt mg/kg	tissue conc contam dry wt mg/kg	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
landfill-field		1000	lettuce		1.3	0.0013	0.05	0.000065	8.125E-05	Samsoe-Petersen et al., 2002
moderate urban poll -field		130	lettuce		0.25	0.0020	0.05	0.0001	0.000125	Samsoe-Petersen et al., 2002
field-wastewater	0.32	2.04	basil	0.18	0.84	0.4100	0.08	0.033	0.04125	Shariatpanahi and Anderson 1986
field-wastewater	0.32	2.04	garden cress	0.16	0.8	0.3900	0.08	0.031	0.03875	Shariatpanahi and Anderson 1986
field-wastewater	0.32	2.04	mint	0.29	0.78	0.3800	0.08	0.031	0.03875	Shariatpanahi and Anderson 1986
field-wastewater	0.32	2.04	tarragon	0.15	0.68	0.3300	0.08	0.027	0.03375	Shariatpanahi and Anderson 1986
flooded gardens		85.2	sorrel		0.99	0.0116	0.08	0.00093	0.001162	Sipter et al. 2008
non-flooded gardens		27.8	sorrel		0.295	0.0106	0.08	0.000849	0.0010612	Sipter et al. 2008
sewage sludge-field			spinach				0.08	0.00048	0.0006	Sridhara Chary et al., 2008
Indust. sewage wastes - field	3.4	183.5	amaranthus	0.12	12.2	0.0660	0.08	0.0054	0.00675	Srikanth et al., 1991
Indust. sewage wastes - field	3.4	183.5	cabbage	0.64	7.52	0.0410	0.078	0.0032	0.004	Srikanth et al., 1991
Indust. sewage wastes - field	3.4	183.5	spinach	0.05	14.94	0.0810	0.086	0.007	0.00875	Srikanth et al., 1991
urban gardens-field-to-grnhs	12	1601	lettuce	2.22	8.67	0.0080	0.045	0.00036	0.00045	Sterrett et al., 1996
field		71.31	Chinese cabbage		0.65	0.0091	0.08	0.000729	0.0009115	Wang et al. 2006
field		71.31	Pakchoi		0.7625	0.0107	0.08	0.000855	0.0010693	Wang et al. 2006
field		71.31	Water spinach		1.2125	0.0170	0.08	0.00136	0.0017003	Wang et al. 2006
field		400.3	Pakchoi		3.28	0.0680	0.08	0.00544	0.0068	Yan et al. 2007
field - smelter	21.6	319.6	leek			0.2760	0.08	0.02208	0.0276	Zheng et al. 2007a
field - smelter		158	Chinese cabbage				0.055	0.018	0.023	Zheng et al. 2007b
field - smelter		297	green onion				0.085	0.006	0.008	Zheng et al. 2007b
field - smelter		297	spinach				0.088	0.025	0.03	Zheng et al. 2007b
field - smelter		139	celery				0.058	0.016	0.02	Zheng et al. 2007b
field - smelter		111	cabbage				0.052	0.019	0.024	Zheng et al. 2007b
field - smelter		111	lettuce				0.042	0.024	0.03	Zheng et al. 2007b
field - smelter		167	mustard				0.071	0.021	0.026	Zheng et al. 2007b

Average lead uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0077±0.0104

Table H.11-2 Lead field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field		12	peach		1.4	0.1167	0.131	0.015283	0.0191042	Basar and Aydmalp (2005)
field		12	peach		2.9	0.2417	0.131	0.031658	0.0395729	Basar and Aydmalp 2005
field		11	peach		0.8	0.0727	0.131	0.009527	0.0119091	Basar and Aydmalp 2005
field		137	cauliflower		2	0.0146	0.126	0.001839	0.0022993	Chumbley and Unwin 1982
indust. sewage-field-Egypt	ND	334	bell pepper		0.4	0.0010	0.074	0.00007	0.0000875	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	cucumber		0.3	0.0009	0.039	0.00004	0.00005	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	fig		0.6	0.0020	0.225	0.00045	0.0005625	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	olive		0.3	0.0009	0.2	0.0002	0.00025	Gorbunov et al., 2003
sewage sludge-field	21.1	67.4	Indian squash	0.33	1.4	0.0200	0.082	0.002	0.0025	Jamali et al., 2007
mining, smelting-field		223.22	capsicum			0.0370	0.074	0.0027	0.003375	Li et al., 2006
mining, smelting-field		223.22	cucumber			0.0460	0.039	0.0018	0.00225	Li et al., 2006
mining, smelting-field		223.22	eggplant			0.0220	0.073	0.0016	0.002	Li et al., 2006
field		14.49	broccoli		0.34	0.0235	0.126	0.002957	0.0036957	Liu et al. 2006
field		14.48	cucumber		1.39	0.0960	0.039	0.003744	0.0046797	Liu et al. 2006
field		14.48	Eggplant		1.3	0.0898	0.073	0.006554	0.0081923	Liu et al. 2006
field		14.48	kidney bean		0.91	0.0628	0.111	0.006976	0.0087198	Liu et al. 2006
field		14.48	pepper		4.25	0.2935	0.126	0.036982	0.0462276	Liu et al. 2006
field		14.47	tomato		5.23	0.3614	0.059	0.021325	0.026656	Liu et al. 2006
air dep, mine waste, poll. Water		751.98	capsicum		4.58	0.0061	0.074	0.00045	0.0005625	Liu et al., 2005
air dep, mine waste, poll. Water	60.49	751.98	string bean	0.84	5.82	0.0077	0.111	0.00086	0.001075	Liu et al., 2005
indust. Poll. Depo. - field		165.85	eggplant		13.15	0.0790	0.073	0.0058	0.00725	Pandey and Pandey, 2009
indust. Poll. Depo. - field		165.85	tomato		15.2	0.0920	0.059	0.0054	0.00675	Pandey and Pandey, 2009
smelter area - urban gardens - field	84	872	tomato	0	1.38	0.0016	0.065	0.0001	0.000125	Pruvot et al., 2006
Kalvebod area		613	blackberry					0.000026	0.0000325	Samsoe-Petersen et al., 2002
Kalvebod area		613	pear					0.000016	0.00002	Samsoe-Petersen et al., 2002
Kalvebod area		613	plum					0.000016	0.00002	Samsoe-Petersen et al., 2002
field-wastewater	0.32	2.04	leek	0.2	0.65	0.3200	0.12	0.038	0.0475	Shariatpanahi and Anderson 1986

Table H.11-2 Lead field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
flooded gardens		85.2	squash		0.673	0.0079	0.082	0.000648	0.0008097	Sipter et al. 2008
flooded gardens		85.2	tomato		0.48	0.0056	0.059	0.000332	0.0004155	Sipter et al. 2008
non-flooded gardens		27.8	squash		0.079	0.0028	0.082	0.000233	0.0002913	Sipter et al. 2008
non-flooded gardens		27.8	tomato		0.083	0.0030	0.059	0.000176	0.0002202	Sipter et al. 2008
smelter contam - field	22	163	tomato		7.15	0.0440	0.065	0.0029	0.003625	Tomov & Alandjiyski, 2006
field		71.31	Eggplant		0.3973	0.0056	0.073	0.000407	0.0005083	Wang et al. 2006
field		71.31	Towel gourd		0.3415	0.0048	0.082	0.000393	0.0004908	Wang et al. 2006
field - smelter	21.6	319.6	aubergine			0.0240	0.066	0.001584	0.00198	Zheng et al. 2007a
field - smelter	21.6	319.6	capsicum			0.0240	0.081	0.001944	0.00243	Zheng et al. 2007a
field - smelter		297	tomato				0.056	0.002	0.003	Zheng et al. 2007b
field - smelter		167	bitter melon				0.066	0.003	0.004	Zheng et al. 2007b

Average lead uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00693±0.0124

Table H.11-3 Lead field studies on protected crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field	50	318.056	corn		1.1	0.0035	0.261	0.000903	0.0011283	Bi et al. 2006
field		156	sweet corn		0.1	0.0006	0.261	0.000167	0.0002091	Chumbley and Unwin 1982
25% mine waste - grnhs	60.9	3600	bush bean	5.53	0	-	0.099	0.00017	0.0002125	Cobb et al., 2000
indust. sewage-field-Egypt	ND	334	bean (spot)		2.2	0.0070	0.894	0.006	0.0075	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	bean (white)		0.9	0.0030	0.894	0.003	0.00375	Gorbunov et al., 2003
sewage sludge-field	21.1	67.4	cluster beans	0.104	0.6	0.0090	0.111	0.001	0.00125	Jamali et al., 2007
sewage sludge-field	21.1	67.4	peas	0.22	0.74	0.0100	0.257	0.003	0.00375	Jamali et al., 2007
mining, smelting-field		223.22	pumpkin			0.0470	0.082	0.0039	0.004875	Li et al., 2006
air dep, mine waste, poll. Water		751.98	corn		1.91	0.0025	0.261	0.00066	0.000825	Liu et al., 2005
field (sewage-fed lake irrigation)			Beans			0.2000	0.111	0.0222	0.02775	Lokeshwari and Chandrappa 2006
smelter area - ag field	30	440	corn	0	0.92	0.0021	0.273	0.00057	0.0007125	Pruvot et al., 2006
Kalvebod area		613	hazelnut					0.00073	0.0009125	Samsoe-Petersen et al., 2002
landfill-field		1000	green bean		1.4	0.0014	0.042	0.00006	0.000075	Samsoe-Petersen et al., 2002
moderate urban poll -field		130	green bean		0.18	0.0010	0.111	0.0002	0.00025	Samsoe-Petersen et al., 2002
sewage sludge-pot-field		154	beans			0.0080	0.222	0.002	0.0025	Sauerbeck, 1991
sewage sludge-pot-field		154	peas			0.0010	0.257	0.0003	0.000375	Sauerbeck, 1991
flooded gardens		85.2	bean		0.26	0.0031	0.111	0.000339	0.0004234	Sipter et al. 2008
non-flooded gardens		27.8	bean		0.141	0.0051	0.111	0.000563	0.0007037	Sipter et al. 2008
field		71.31	Cowpea		0.2023	0.0028	0.257	0.000729	0.0009115	Wang et al. 2006
field - smelter	21.6	319.6	kidney bean			0.0320	0.103	0.003296	0.00412	Zheng et al. 2007a
field - smelter		297	cowpea				0.097	0.003	0.004	Zheng et al. 2007b
field - smelter		297	pumpkin				0.065	0.001	0.001	Zheng et al. 2007b

Average lead uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00282±0.00565

Table H.11-4 Lead field studies on root crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-ground water		28	potato		0.5	0.0179	0.222	0.003974	0.0049673	Alam et al. 2003
salt	40.5	744.5	carrot	0.312	5.754	0.0077	0.118	0.000912	0.00114	Alexander et al. (2006)
salt	40.5	744.5	Onion	1.418	7.458	0.0100	0.125	0.001252	0.0015652	Alexander et al. 2006
smelter - field - home gardens		130	carrot		2.2	0.0169	0.118	0.002	0.0025	Chaney et al., 1988
smelter - field - home gardens		48	potato		2.6	0.0542	0.202	0.01	0.0125	Chaney et al., 1988
field		103	beetroot		0.4	0.0039	0.222	0.000862	0.0010777	Chumbley and Unwin 1982
field		97	leeks		0.8	0.0082	0.2	0.001649	0.0020619	Chumbley and Unwin 1982
field		176	potato		0.2	0.0011	0.222	0.000252	0.0003153	Chumbley and Unwin 1982
field		110	radish		2.9	0.0264	0.222	0.005853	0.0073159	Chumbley and Unwin 1982
field		107	onions		0.6	0.0056	0.125	0.000701	0.0008762	Chumbley and Unwin 1982
25% mine waste - grnhs	60.9	3600	radish	0	92.4	0.0257	0.047	0.0012	0.0015	Cobb et al., 2000
smelter flue-dust	6.8	146.3	potato	0.2	0.2	0.0014	0.222	0.000303	0.0003794	Dudka et al. (1996)
smelter flue-dust	6.8	340	potato	0.2	0.4	0.0012	0.222	0.000261	0.0003265	Dudka et al. 1996
smelter flue-dust	6.8	2202.5	potato	0.2	0.7	0.0003	0.222	7.06E-05	8.82E-05	Dudka et al. 1996
smelter flue-dust	6.8	5452.5	potato	0.2	0.9	0.0002	0.222	3.66E-05	4.58E-05	Dudka et al. 1996
urban gardens-field			carrot				0.118	0.0006	0.00075	Finster et al., (2004)
urban gardens-field			onion				0.125	0.004	0.005	Finster et al., 2004
urban gardens-field			radish				0.047	0.00094	0.001175	Finster et al., 2004
indust. sewage-field-Egypt	ND	334	garlic		1	0.0030	0.387	0.001	0.00125	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	onion		1.1	0.0030	0.125	0.0004	0.0005	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	radish		2.3	0.0070	0.047	0.0003	0.000375	Gorbunov et al., 2003
sewage sludge-field	70	259	carrot	0.33	0.48	0.0040	0.118	0.0005	0.000625	Hooda et al., 1997
Pb arsenate - grnhs	60.9	342.3	carrot	3.9	13.3	0.0400	0.118	0.005	0.00625	Hutchinson et al. (1974)
Pb arsenate - grnhs	60.9	342.3	onion	10	75.4	0.2000	0.125	0.03	0.0375	Hutchinson et al. 1974
Pb arsenate - grnhs	60.9	342.3	parsnip	7.8	14.8	0.0400	0.209	0.008	0.01	Hutchinson et al. 1974
Pb arsenate - grnhs	60.9	342.3	radish	7.9	27.5	0.0800	0.047	0.004	0.005	Hutchinson et al. 1974
field		14.49	carrot		0.92	0.0635	0.118	0.007492	0.0093651	Liu et al. 2006
field		14.49	leek		0.92	0.0635	0.146	0.00927	0.0115873	Liu et al. 2006
field		14.48	radish		0.47	0.0325	0.047	0.001526	0.0019069	Liu et al. 2006
Env. contam. Soil (paint?) - potted - grnhs		2000	beet		19	0.0095	0.127	0.001	0.00125	Nicklow et al., 1983

Table H.11-4 Lead field studies on root crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
Env. contam. Soil (paint?) - potted - grnhs		2000	carrot		34	0.0170	0.118	0.002	0.0025	Nicklow et al., 1983
Env. contam. Soil (paint?) - potted - grnhs		2000	turnip		22	0.0110	0.085	0.0009	0.001125	Nicklow et al., 1983
indust. Poll. Depo. - field		165.85	carrot		8.16	0.0490	0.118	0.0058	0.00725	Pandey and Pandey, 2009
indust. Poll. Depo. - field		165.85	radish		11.7	0.0710	0.047	0.0033	0.004125	Pandey and Pandey, 2009
smelter area - ag field	30	440	potato	0.099	0.099	0.0002	0.202	0.000045	5.625E-05	Pruvot et al., 2006
smelter area - urban gardens - field	84	872	carrot	0.25	1.17	0.0013	0.118	0.00024	0.0003	Pruvot et al., 2006
smelter area - urban gardens - field	84	872	leek	0.34	2.67	0.0031	0.146	0.00045	0.0005625	Pruvot et al., 2006
smelter area - urban gardens - field	84	872	potato	0	0.15	0.0002	0.202	0.000034	0.0000425	Pruvot et al., 2006
smelter area - urban gardens - field	84	872	radish	0	3.83	0.0044	0.047	0.00021	0.0002625	Pruvot et al., 2006
landfill-field		1000	carrot unp		5.1	0.0051	0.104	0.00053	0.0006625	Samsoe-Petersen et al., 2002
landfill-field		1000	potato unp		2	0.0020	0.113	0.00023	0.0002875	Samsoe-Petersen et al., 2002
landfill-field		1000	radish		7.4	0.0074	0.036	0.00027	0.0003375	Samsoe-Petersen et al., 2002
moderate urban poll -field		130	carrot unp		0.93	0.0070	0.118	0.0009	0.001125	Samsoe-Petersen et al., 2002
moderate urban poll -field		130	potato unp		0.18	0.0010	0.222	0.0003	0.000375	Samsoe-Petersen et al., 2002
moderate urban poll -field		130	radish		1.65	0.0100	0.085	0.001	0.00125	Samsoe-Petersen et al., 2002
sewage sludge-pot-field		154	carrots			0.0030	0.118	0.0004	0.0005	Sauerbeck, 1991
sewage sludge-pot-field		154	radish			0.0200	0.05	0.0009	0.001125	Sauerbeck, 1991
field-wastewater	0.32	2.04	onion	0.22	0.46	0.2300	0.125	0.028	0.035	Shariatpanahi and Anderson 1986
field-wastewater	0.32	2.04	radish	0.28	0.73	0.3600	0.047	0.02	0.025	Shariatpanahi and Anderson 1986
flooded gardens		85.2	carrot		0.81	0.0095	0.118	0.001122	0.0014023	Sipter et al. 2008
flooded gardens		85.2	onion		1.06	0.0124	0.125	0.001555	0.001944	Sipter et al. 2008
non-flooded gardens		27.8	carrot		0.278	0.0100	0.118	0.00118	0.001475	Sipter et al. 2008
non-flooded gardens		27.8	onion		0.13	0.0047	0.125	0.000585	0.0007307	Sipter et al. 2008
smelter contam - field	22	163	potato		2.95	0.0180	0.202	0.0037	0.004625	Tomov & Alandjiyski, 2006
field - smelter	21.6	319.6	carrot			0.0320	0.108	0.003456	0.00432	Zheng et al. 2007a
field - smelter	21.6	319.6	turnip			0.0270	0.088	0.002376	0.00297	Zheng et al. 2007a
field - smelter		167	potato				0.11	0.001	0.001	Zheng et al. 2007b

Average lead uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00403±0.0075

Table H.12-1 Mercury field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
Hgt pots -env. chamber		17.6	cabbage		1.5	0.09	0.08	0.007	0.00875	Caille (2005)
Hgt pots -env. chamber		17.6	rape		1.7	0.09	0.08	0.008	0.01	Caille et al., 2005
field-compost			lettuce				0.05	0.0122355	0.0152944	Cappon 1987
field-compost			spinach				0.08	0.0137064	0.017133	Cappon 1987
field-compost			Swiss chard				0.08	0.01201	0.0150125	Cappon 1987
field		4.77	amaranth		0.27	0.0566038	0.08	0.0045283	0.0056604	Liu et al. 2006
field		4.77	cabbage		0.21	0.0440252	0.08	0.003522	0.0044025	Liu et al. 2006
field		4.77	celery		0.31	0.0649895	0.08	0.0051992	0.006499	Liu et al. 2006
field		4.77	Ch cabbage		0.15	0.0314465	0.08	0.0025157	0.0031447	Liu et al. 2006
field		4.77	Ch chive		0.32	0.067086	0.08	0.0053669	0.0067086	Liu et al. 2006
field		5.5	leek		0.19	0.0345455	0.08	0.0027636	0.0034545	Liu et al. 2006
field		4.77	pakchoi		0.41	0.0859539	0.08	0.0068763	0.0085954	Liu et al. 2006
field-contam fungicide -greenhouse grown	ND	1.64	lettuce		0.173	0.10549	0.05	0.0052745	0.0065931	(MacLean, 1974)
field-contam fungicide -greenhouse grown	ND	7.13	lettuce		0.103	0.01445	0.05	0.0007225	0.0009031	MacLean 1974
sewage sludge - field		2.5	cabbage		0.01	0.004	0.08	0.0003	0.000375	Muntau et al., 1987
field-wastewater	0.06	0.16	basil	0.05	0.08	0.5	0.08	0.04	0.05	Shariatpanahi and Anderson 1986
field-wastewater	0.06	0.16	gard cress	0.04	0.12	0.75	0.08	0.06	0.075	Shariatpanahi and Anderson 1986
field-wastewater	0.06	0.16	mint	0.06	0.08	0.5	0.08	0.04	0.05	Shariatpanahi and Anderson 1986
field-wastewater	0.06	0.16	tarragon	0.04	0.13	0.81	0.08	0.065	0.08125	Shariatpanahi and Anderson 1986
flooded gardens		0.81	sorrel		0.06	0.0740741	0.08	0.0059259	0.0074074	Sipter et al. 2008
field - smelter	0.037	1.28	leek			0.139	0.08	0.01112	0.0139	Zheng et al. 2007a
field - smelter	0.037	0.76	Ch cabbage				0.055	0.016	0.02	Zheng et al. 2007a
field - smelter	0.037	1.5	Grn onion				0.085	0.01	0.0125	Zheng et al. 2007a
field - smelter	0.037	1.5	spinach				0.088	0.005	0.00625	Zheng et al. 2007a
field - smelter	0.037	0.4	celery				0.058	0.01	0.0125	Zheng et al. 2007a
field - smelter	0.037	0.5	cabbage				0.052	0.031	0.03875	Zheng et al. 2007a
field - smelter	0.037	0.5	lettuce				0.042	0.015	0.01875	Zheng et al. 2007a
field - smelter	0.037	0.3	mustard				0.071	0.01	0.0125	Zheng et al. 2007a

Average mercury uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0163±0.0202

Table H.12-2 Mercury field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field survey			peppers			0.00222	0.126	0.0002797	0.0003497	Cambra et al. 1999
field-compost			broccoli				0.126	0.0145385	0.0181731	Cappon 1987
field-compost			cabbage				0.08	0.0120093	0.0150117	Cappon 1987
field-compost			cucmber				0.039	0.0002636	0.0003295	Cappon 1987
field-compost			pepper				0.074	0.0014145	0.0017681	Cappon 1987
field-compost			squash				0.082	0.0016629	0.0020787	Cappon 1987
field-compost			tomato				0.059	0.0036445	0.0045557	Cappon 1987
field		5.5	broccoli		0.12	0.0218182	0.126	0.0027491	0.0034364	Liu et al. 2006
field		4.03	cucumber		0.15	0.0372208	0.039	0.0014516	0.0018145	Liu et al. 2006
field		4.77	Eggplant		0.26	0.0545073	0.073	0.003979	0.0049738	Liu et al. 2006
field		4.77	kidney bean		0.27	0.0566038	0.111	0.006283	0.0078538	Liu et al. 2006
field		4.77	pepper		0.14	0.0293501	0.126	0.0036981	0.0046226	Liu et al. 2006
field		4.77	tomato		0.13	0.0272537	0.059	0.001608	0.00201	Liu et al. 2006
pots - phenyl mercuric acetate	0.08	5.24	tomato	0.034	0.037	0.0071	0.059	0.00042	0.000525	MacLean 1974
field-wastewater	0.06	0.16	leek	0.04	0.1	0.63	0.12	0.075	0.09375	Shariatpanahi and Anderson 1986
flooded gardens		0.81	squash		0.037	0.045679	0.082	0.0037457	0.0046821	Sipter et al. 2008
flooded gardens		0.81	tomato		0.01	0.0123457	0.059	0.0007284	0.0009105	Sipter et al. 2008
field - smelter	0.037	1.28	aubergine			0.003	0.066	0.000198	0.0002475	Zheng et al. 2007a
field - smelter	0.037	1.28	capsicum			0.007	0.081	0.000567	0.0007088	Zheng et al. 2007a
field - smelter	0.037	1.5	tomato				0.056	0.004	0.005	Zheng et al. 2007a
field - smelter	0.037	0.3	bitter melon				0.066	0.016	0.02	Zheng et al. 2007a

Average mercury uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00855±0.0194

Table H.12-3 Mercury field studies on protected crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field survey			broad beans			0.003506	0.126	0.0004418	0.0005522	Cambra et al. 1999
field-compost			bean				0.111	0.0011126	0.0013907	Cappon 1987
field	0.15	0.38	corn		0.011	0.0289474	0.261	0.0075553	0.0094441	Feng et al. (2006)
Hgt field-smelter-9 sites			brown rice			0.002	0.888	0.002	0.0025	Horvet et al., 2003
Hgt field-smelter-2 sites			brown rice			0.0001	0.888	0.00009	0.0001125	Horvet et al., 2003
Hgt field-clean area-2 sites			brown rice			0.009	0.888	0.008	0.01	Horvet et al., 2003
field		0.21	wheat		0.003	0.0142857	0.875	0.0125	0.015625	Huang et al. (2008)
HgCl2 - pots - chamber	ND		oats	0.009	0.013	0.002	0.917	0.0018	0.00225	John 1972
HgCl2 - pots - chamber	ND		peas	0.001	0.002	0.00033	0.257	0.000085	0.0001063	John 1972
Hgt field-smelter-23 sites		0.1782	corn		0.0061	0.03	0.261	0.0089	0.011125	Li et al., (2008)
pots - phenyl mercuric acetate	0.08	5.24	oats	0.113	0.163	0.031	0.917	0.029	0.03625	MacLean 1974
pots - phenyl mercuric acetate	0.08	5.24	soybeans	0.074	0.076	0.015	0.925	0.013	0.01625	MacLean 1974
flooded gardens		0.81	bean		0.03	0.037037	0.111	0.0041111	0.0051389	Sipter et al. 2008
field - smelter	0.037	1.28	kidney bean			0.067	0.103	0.006901	0.0086263	Zheng et al. 2007a
field - smelter	0.037	1.5	cowpea				0.097	0.001	0.00125	Zheng et al. 2007a

Average mercury uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00804±0.0096

Table H.12-4 Mercury field studies on root crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-compost			Beet				0.164	0.0104746	0.0130932	Cappon 1987
field-compost			carrot				0.118	0.0036308	0.0045385	Cappon 1987
field-compost			onion				0.125	0.0105478	0.0131847	Cappon 1987
field-compost			radish				0.222	0.0129371	0.0161713	Cappon 1987
field-compost			turnip				0.222	0.0056406	0.0070507	Cappon 1987
HgCl2 - pots - chamber	ND		carrot	0.044	0.053	0.0075	0.118	0.00089	0.0011125	John (1972)
HgCl2 - pots - chamber	ND		radish	0.013	0.026	0.02	0.085	0.0017	0.002125	John 1972
field		5.5	carrot		0.24	0.0436364	0.118	0.0051491	0.0064364	Liu et al. 2006
field		4.77	radish		0.21	0.0440252	0.2	0.008805	0.0110063	Liu et al. 2006
pots - phenyl mercuric acetate	0.08	5.24	carrot	0.086	0.18	0.034	0.118	0.0041	0.005125	MacLean 1974
pots - phenyl mercuric acetate	0.08	5.24	potato	0.047	0.055	0.01	0.222	0.0023	0.002875	MacLean 1974
field-wastewater	0.06	0.16	onion	0.06	0.06	0.38	0.125	0.047	0.05875	Shariatpanahi and Anderson 1986
field-wastewater	0.06	0.16	radish	0.04	0.08	0.5	0.085	0.043	0.05375	Shariatpanahi and Anderson 1986
flooded gardens		0.81	carrot		0.02	0.0246914	0.118	0.0029136	0.003642	Sipter et al. 2008
flooded gardens		0.81	onion		0.02	0.0246914	0.125	0.0030864	0.003858	Sipter et al. 2008
field - smelter	0.037	1.28	carrot			0.044	0.108	0.004752	0.00594	Zheng et al. 2007a
field - smelter	0.037	1.28	turnip			0.034	0.088	0.002992	0.00374	Zheng et al. 2007a
field - smelter	0.037	0.3	potato				0.11	0.002	0.0025	Zheng et al. (2007b)

Average mercury uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0119±0.0167

Table H.13-1 Nickel field studies on leafy crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field (industrial sewage irrigation)		13.37	palak (spinach)		4.2	0.31	0.08	0.02	0.025	Kumar Sharma et al., 2007
field (industrial sewage irrigation)		15.61	palak (spinach)		5.9	0.38	0.08	0.03	0.0375	Kumar Sharma et al., 2007
field (industrial sewage irrigation)		14.52	palak (spinach)		2.6	0.18	0.08	0.02	0.025	Kumar Sharma et al., 2007
indust. Poll. Depo. - field		119.32	amaranthus		9.5	0.08	0.08	0.0064	0.008	Pandey and Pandey, 2009
indust. Poll. Depo. - field		119.32	spinach		10.62	0.089	0.08	0.0071	0.008875	Pandey and Pandey, 2009
landfill-field		49	lettuce		1.23	0.025	0.05	0.00125	0.0015625	Samsoe-Petersen et al., 2002
sewage sludge - field		120	cabbage		24	0.2	0.08	0.02	0.025	Muntau et al., 1987
sewage sludge-field	22.5	51.8	spinach	4.76	9.46	0.178	0.08	0.014	0.0175	Hooda et al., 1997
sewage sludge-field	28.1	34.6	spinach	0.88	1.2	0.03	0.08	0.003	0.00375	Jamali et al., 2007
sewage sludge-field			spinach				0.08	0.0048	0.006	Sridhara Chary et al., (2008)
urban gardens-field-to-greenhouse	10	50.7	lettuce	0.73	1.25	0.024	0.045	0.00108	0.00135	Sterrett et al., 1996

Average nickel uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0145±0.0121

Table H.13-2 Nickel field studies on exposed crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field		112	peach		1.5	0.0133929	0.131	0.0017545	0.0021931	Basar and Aydmalp 2005
field		117	peach		1.6	0.0136752	0.131	0.0017915	0.0022393	Basar and Aydmalp 2005
field		122	peach		2	0.0163934	0.131	0.0021475	0.0026844	Basar and Aydmalp 2005
highly contam area		53	blackberry					0.0021	0.002625	Samsoe-Petersen et al., 2002
highly contam area		53	pear					0.0013	0.001625	Samsoe-Petersen et al., 2002
highly contam area		53	plum					0.0007	0.000875	Samsoe-Petersen et al., 2002
indust. Poll. Depo. - field		119.32	eggplant		7.92	0.066	0.073	0.0048	0.006	Pandey and Pandey, 2009
indust. Poll. Depo. - field		119.32	tomato		9.85	0.083	0.059	0.0049	0.006125	Pandey and Pandey, 2009
indust. sewage-field-Egypt	ND	106	bell pepper		0.7	0.007	0.074	0.0005	0.000625	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	cucumber		0.43	0.004	0.039	0.0002	0.00025	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	fig		1.6	0.02	0.225	0.0045	0.005625	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	olive		0.41	0.004	0.2	0.0008	0.001	Gorbunov et al., 2003
sewage sludge-field	28.1	34.6	Indian squash	1.3	2.1	0.06	0.082	0.005	0.00625	Jamali et al., 2007

Average nickel uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00293±0.00226

Table H.13-3 Nickel field studies on protected crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field (sewage-fed lake irrigation)			Beans			0.1	0.111	0.0111	0.013875	Lokeshwari and Chandrappa (2006)
highly contam area		53	hazelnut					0.033	0.04125	Samsoe-Petersen et al., 2002
indust. sewage-field-Egypt	ND	106	bean (spot)		6.9	0.07	0.894	0.06	0.075	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	bean (white)		1.9	0.02	0.894	0.02	0.025	Gorbunov et al., 2003
landfill-field		49	green bean		6.37	0.13	0.076	0.0099	0.012375	Samsoe-Petersen et al., 2002
sewage sludge-field	28.1	34.6	cluster beans	1.21	2.1	0.06	0.111	0.007	0.00875	Jamali et al., 2007
sewage sludge-field	28.1	34.6	peas	1.12	1.18	0.03	0.257	0.009	0.01125	Jamali et al., 2007
sewage sludge-pot-field		25	beans			0.3	0.099	0.03	0.0375	Sauerbeck, 1991
sewage sludge-pot-field		25	peas			0.2	0.257	0.04	0.05	Sauerbeck, 1991

Average nickel uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0306±0.0224

Table H.13-4 Nickel field studies on root crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
indust. Poll. Depo. - field		119.32	carrot		3.65	0.031	0.118	0.0037	0.004625	Pandey and Pandey, 2009
indust. Poll. Depo. - field		119.32	radish		3.98	0.033	0.047	0.0016	0.002	Pandey and Pandey, 2009
indust. sewage-field-Egypt	ND	106	garlic		2.6	0.02	0.125	0.003	0.00375	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	onion		3.1	0.03	0.125	0.004	0.005	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	radish		3.8	0.04	0.085	0.003	0.00375	Gorbunov et al., 2003
landfill-field		49	carrot (unpeeled)		1.86	0.038	0.132	0.005	0.00625	Samsoe-Petersen et al., 2002
landfill-field		49	potato (unpeeled)		0.34	0.007	0.185	0.0013	0.001625	Samsoe-Petersen et al., 2002
landfill-field		49	radish		1.57	0.032	0.048	0.0015	0.001875	Samsoe-Petersen et al., 2002
sewage sludge-field	22.5	51.8	carrot	2.17	5.28	0.118	0.118	0.014	0.0175	Hooda et al., (1997)
sewage sludge-pot-field		25	carrots			0.08	0.118	0.009	0.01125	Sauerbeck, 1991
sewage sludge-pot-field		25	radish			0.2	0.05	0.01	0.0125	Sauerbeck, 1991

Average nickel uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00638±0.00516

Table H.15-1 Selenium field studies on leafy crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-fly ash	1.5	1.7	cabbage	0.07	0.2	0.1	0.08	0.009	0.01125	Furr et al. 1978
sewage sludge - field		0.4	cabbage		1.1	2.8	0.08	0.2	0.25	Muntau et al., 1987
field-compost			lettuce				0.05	0.008482	0.0106025	Cappon 1987
field-compost			lettuce				0.05	0.010372	0.012965	Cappon 1987
field		9.84	lettuce		19.16	1.94715	0.05	0.0973575	0.1216969	van Mantgem et al. (1996)
field		6.18	lettuce		5.61	0.90777	0.05	0.0453885	0.0567356	van Mantgem et al. 1996
field		15.9	lettuce		13.63	0.85723	0.05	0.0428615	0.0535769	van Mantgem et al. 1996
field		16.83	lettuce		27.9	1.65775	0.05	0.0828875	0.1036094	van Mantgem et al. 1996
field		17.37	lettuce		12.37	0.71215	0.05	0.0356075	0.0445094	van Mantgem et al. 1996
field-compost			spinach				0.08	0.016888	0.02111	Cappon 1987
field-compost			Swiss chard				0.08	0.00957	0.0119625	Cappon 1987

Average selenium uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0587±0.0713

Table H.15-2 Selenium field studies on exposed crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-fly ash-potted soil	0.3	1.2	apple (w/o seeds)	0.01	0.03	0.03	0.159	0.004	0.005	Furr et al. (1979)
field-compost			broccoli				0.126	0.0130125	0.0162656	Cappon 1987
field-fly ash-potted soil	0.3	1.2	cabbage	0.04	2.4	2	0.08	0.2	0.25	Furr et al. 1979
field-compost			cabbage				0.08	0.0216667	0.0270833	Cappon 1987
field-compost			cucmber				0.039	0.0010563	0.0013203	Cappon 1987
field-compost			pepper				0.074	0.0025107	0.0031384	Cappon (1987)
field-compost			squash				0.082	0.0027089	0.0033862	Cappon 1987
field-fly ash-potted soil	0.3	1.2	tomato	0.015	1.5	1.2	0.059	0.07	0.0875	Furr et al. 1979
field-compost			tomato				0.059	0.0099387	0.0124234	Cappon 1987
field-fly ash - pot	1.5	1.7	tomato	0.01	0.02	0.01	0.059	0.007	0.00875	Furr et al. 1978

Average selenium uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0415±0.0776

Table H.15-3 Selenium field studies on protected crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-compost			bean				0.111	0.0070366	0.0087958	Cappon 1987
field-smelter		16.9	brown rice		1.06	0.06	0.888	0.056	0.07	Horvet et al., (2003)
field-fly ash - pot	1.5	1.7	bush bean	0.02	0.07	0.04	0.111	0.005	0.00625	Furr et al. 1978
field-fly ash-potted soil	0.3	1.2	bush bean	0.025	1.3	1.1	0.111	0.1	0.125	Furr et al. 1979
field-fly ash - pot	1.5	1.7	corn	0.02	0.05	0.03	0.895	0.03	0.0375	Furr et al. 1978
field-fly ash-potted soil	0.3	1.2	Japanese millet grain	0.025	1.4	1.1	0.888	1	1.25	Furr et al. 1979
field-fly ash-potted soil			onion		2.3	1.9	0.125	0.2375	0.296875	Furr et al. 1979

Average selenium uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.256±0.450

Table H.15-4 Selenium field studies on root crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-compost			Beet				0.164	0.0098107	0.0122634	Cappon 1987
field-fly ash-potted soil	0.3	1.2	carrot	0.015	1.5	1.3	0.118	0.1	0.125	Furr et al. 1979
field-compost			carrot				0.118	0.0082179	0.0102723	Cappon 1987
field-fly ash - pot	1.5	1.7	carrot (peeled)	0.02	0.06	0.04	0.118	0.004	0.005	Furr et al. 1978
field-compost			onion				0.125	0.0550223	0.0687779	Cappon 1987
field-fly ash - pot	1.5	1.7	Onion (peeled)	0.02	0.21	0.1	0.125	0.02	0.025	Furr et al. 1978
field-fly ash-potted soil	0.3	1.2	potato	0.025	1.8	1.5	0.222	0.3	0.375	Furr et al. 1979
field-fly ash - pot	1.5	1.7	Potato (peeled)	0.02	0.03	0.02	0.222	0.004	0.005	Furr et al. (1978b)
field-compost			radish				0.222	0.0391143	0.0488929	Cappon 1987
field-compost			turnip				0.222	0.0112321	0.0140402	Cappon 1987

Average selenium uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0689±0.114

H.11 Summary and Recommendations

OEHHA recommends the root uptake factors in Table H.16 for metals and metalloids.

Table H.16 Recommended Soil-to-plant uptake factors for inorganic metals and metalloids in edible crops[a]

Element	Leafy	Exposed	Protected	Root
Arsenic	1×10^{-2}	2×10^{-2}	7×10^{-2}	8×10^{-3}
Beryllium	2×10^{-4}	8×10^{-3}	3×10^{-4}	5×10^{-3}
Cadmium	1×10^{-1}	2×10^{-2}	1×10^{-2}	8×10^{-2}
Chromium (VI)	3×10^{-1}	2×10^{-2}	7×10^{-2}	3×10^{0}
Fluoride	4×10^{-2}	4×10^{-3}	4×10^{-3}	9×10^{-3}
Lead	8×10^{-3}	7×10^{-3}	3×10^{-3}	4×10^{-3}
Mercury	2×10^{-2}	9×10^{-3}	1×10^{-2}	2×10^{-2}
Nickel	1×10^{-2}	3×10^{-3}	3×10^{-2}	6×10^{-3}
Selenium	6×10^{-2}	4×10^{-2}	3×10^{-1}	7×10^{-2}

[a] Soil-to-plant UFs represent the fresh weight concentration of a contaminant in the plant part over the wet weight concentration of contaminant in the soil.

H.12 References

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Appendix I

Fish Bioaccumulation Factors

I.1 Introduction

The algorithm used in the AB-2588 risk assessment to estimate exposure to contaminants via intake of angler-caught fish contains a chemical-specific variable known as a bioaccumulation factor (BAF). Fish are exposed to chemicals that are deposited into their aqueous environment from airborne sources. Only a small subset of Hot Spots chemicals are wholly or partially in the particulate phase and thus subject to deposition. These chemicals include semivolatile organic chemicals and toxic metals. Table I-1 presents the chemical-specific BAF values derived by OEHHA for the Hot Spots program. This appendix outlines the methods used for estimating BAFs and summarizes the available literature used for deriving the chemical-specific BAFs recommended in Table I-1.

Table I-1. Recommended Default Fish BAFs for Edible (Muscle) Tissue[a]

Organic Chemicals[b]	
Diethylhexylphthalate (DEHP)	40
Hexachlorobenzene (HCB)	80,000
Hexachlorocylcohexanes (HCH)	3000
Polycyclic aromatic hydrocarbons (PAH)	800
Polychlorinated biphenyls (PCB)	2,000,000
Polychlorinated dibenzo-p-dioxins and furans (PCDD/F)	300,000
Inorganic and Organic Metals[c]	
Arsenic	20
Beryllium	40
Cadmium	40
Chromium	20
Lead	20
Mercury Inorganic mercury Methylmercury	 80 6,000,000
Nickel	20
Selenium	1000

[a] All BAFs were rounded to the nearest whole number.

[b] Lipid-normalized to adult rainbow trout with 4% lipid content in muscle tissue, and based on the freely dissolved fraction of organic chemical in water under conditions of average POC and DOC in U.S. lakes and reservoirs.

[c] Based on wet weight muscle tissue concentration, and on the total water concentration of the metal or metalloid in water, with the exception of methyl mercury, which assumes a translator of 3.2% for freely dissolved MeHg in water compared to the total Hg water concentration.

Accumulation of a chemical in fish is a physical-chemical process by which chemicals tend to apportion themselves between the fish and the fish's contact with its environment. The environment in this case is defined broadly to include the water, food that the fish eats, and contact with materials other than water. Accumulation of chemicals in fish may result in human exposure from fish consumption, which may be significant relative to other exposure pathways considered in the Hot Spots Program.

The Hot Spots program previously only considered the physical-chemical transfer of chemicals from the water column to the fish. This approach does not address other potentially important sources of toxic contaminant contributions to fish and can thus underestimate human exposure for some chemicals. This issue is discussed in more detail below.

The BAF reflects the uptake and retention of a chemical by fish from all surrounding media (e.g., water, food, sediment) when a steady-state concentration has been reached between the fish and the media. The BAF will vary depending on the organ or tissue of interest, but is also often expressed as the chemical accumulation in the whole fish. The BAF is defined under the Hot Spots program as representing the ratio of a concentration of a chemical in edible tissue, specifically the whole muscle tissue or muscle lipid fraction, to its concentration in the surrounding water in situations where the organism and its food are exposed and the ratio does not change substantially over time. The BAF is calculated as:

$$BAF = C_t / C_w \qquad \text{Eq. I.1}$$

where:
C_t = concentration of the chemical in wet tissue
C_w = concentration of chemical in water

Lipophilic, organic chemicals tend to concentrate in the lipid fraction of fish and the resulting BAF is often lipid normalized to express the concentration of chemical in lipid (see below). The concentration of a chemical in water is often expressed in milligrams or micrograms of chemical per liter of water (i.e., mg/L or µg/L) and the concentration in tissue is often expressed in µg of chemical per kg tissue (µg/kg, or ppb). The BAF can be represented as a unitless factor through conversion of a volume of water to a mass (1 L water ≈ 1 kg), or simply represented in L/kg.

In some instances, the BAF may be based on a bioconcentration factor (BCF). The BCF is defined as representing the ratio of a concentration of a chemical in tissue to its concentration in the surrounding water only when a steady-state concentration has been reached between the two media. Potential fish exposure via food sources is not included. Laboratory accumulation studies often determine BCFs due to the simplicity of the test and easier comparison with other BCF studies. Currently, U.S. EPA (2003a) recommends use of BCFs only for exposure to inorganic metals, presumably because intake of inorganic metals by fish via food sources is minor compared to uptake from water. However, a review of the literature by OEHHA suggests contaminated food sources can also be an important source of metal accumulation in fish tissues. Thus, reliance on BCFs to estimate fish exposure may also underestimate the actual accumulation of a metal in fish.

For semi- or non-volatile organic chemicals that are highly persistent and hydrophobic (generally with a log K_{ow}>4), the magnitude of bioaccumulation by fish via food sources

can be substantially greater than the magnitude of bioaccumulation via exposure to water. For such chemicals, only true BAFs adequately assess accumulation of the chemical in fish tissues. For many of these persistent organic chemicals, biomagnification can occur. Biomagnification is the process through which chemical concentrations in fish increase as the chemical moves up the food chain, essentially through food sources. This process occurs because there are fewer organisms feeding off of more organisms at each level in the food chain, thus concentrating the chemical contaminants.

Numerous variables can affect uptake of persistent organic chemicals and inorganic metals in fish, therefore literature sources that reflected potential chemical accumulation as might occur under the "Hot Spots" program were our primary focus. That is, BCF/BAFs were primarily based on the edible portion (i.e., muscle tissue) of freshwater sport fish common to California lentic environments. Lentic environments consist mainly of standing water bodies including lakes, reservoirs and ponds. Sport fish that are caught and consumed in California are predominantly in trophic levels 3 and 4. These fish are typically of highest economic value and include predatory and carnivorous fish that feed on lower trophic level animals. BAF values for trophic level 2 organisms (e.g., zooplankton and larval fish stages) and non-sport fish, such as mosquito fish and the fathead minnow, were not considered unless there was a lack of accumulation data for higher trophic level sport fish.

The muscle tissue is defined here as the edible tissue of fish, although some ethnic groups may also eat various organs of fish. OEHHA's California fish advisories recommend against eating the liver and other organs of fish, because they may have higher concentrations of organic contaminants than the muscle tissue (OEHHA, 2003). In addition, most inorganic metals will also concentrate in the organs, particularly the kidney and liver. Thus, the BAFs derived in this document cannot be used for estimating accumulation of chemicals in organs other than muscle tissue, as doing so could seriously underestimate the dose received by consuming fish organs and tissues other than muscle.

In California, common freshwater sport fish caught for consumption include various species of trout, catfish, bass, perch, sunfish and carp (CDFG, 2007). Mean muscle lipid content and trophic level data for some sport-fish are shown in Table I-2. In general, the size of the sport fish should be representative of the size being consumed by the target human population. Thus, the mean values are based on fish sizes that are caught and consumed by anglers. As Table I-2 shows, both muscle lipid content and trophic level can increase with increasing length (and age) of the fish. In some instances, lipid content or trophic level based on fish length, in cm, is provided.

Table I-2. Percent Muscle Lipid Content and/or Mean Trophic Level for some Freshwater Sport-Fish Found in California

Common Name	Mean % Muscle Lipid	Mean Trophic Level
Carp (*Cypinus carpio*)	4.45	3 (10-23 cm) 2.4 (>23 cm)
Catfish		
Black bullhead	1.12	3
Brown bullhead	2.79	3
Channel catfish	5.00	3.1 (5-30 cm) 2.8-4 (36-54 cm)
White catfish	2.15	
Yellow catfish	0.75	
Blue catfish		3
Flathead catfish		3.8
Perch		
Yellow perch	0.66	3.4
Trout		
Rainbow trout	4.00	3 (<30 cm) 3.6 (30-50 cm) 4 (>50 cm)
Brook trout	1.51	3.2
Brown trout	3.81	
Cutthroat trout	1.23	3 (<40 cm) 3.2 (>40 cm)
Lake trout	10.90	3.7 (20-30 cm) 3.9 (30-40 cm) 4.2 (>40 cm)
Bass		
Smallmouth	1.1	
Largemouth	1.03 (35-48 cm) 3.1 (54 cm)	
Black crappie	0.57 (14-23 cm)	

Sources: U.S. EPA (1998); OEHHA (1999); SFBRWQCB (2005); Morrison et al. (1997)

I.1.1 Uptake and Accumulation of Semi- or Non-Volatile Organic Chemicals in Fish Tissues

Much of the field data for BAFs of organic chemicals comes from studies in the Great Lakes region (Eisenreich et al., 1981). The large surface area of the lakes, long hydraulic residence times, and major pollution sources near and upwind of the lakes have a significant impact on airborne deposited trace organic inputs.

For lipophilic, bioaccumulative organic chemicals, U.S. EPA (1998) recommends calculating a BAF based on the concentration of freely dissolved chemical in the ambient water and the lipid-normalized concentration in tissue. Regarding lipid

normalization, the BAF of lipophilic organic chemicals is usually directly proportional to the percent lipid content in the tissue of interest (U.S. EPA, 1998). For example, a fish with four percent lipid content would accumulate twice the amount of a chemical as a fish with two percent lipid content, all else being equal. Normalizing BAFs or BCFs to lipid content allows comparison between different fish species on the basis of factors other than percent lipid content. The lipid-normalized concentration is expressed as:

$$C = C_t / f \qquad \text{Eq. I.2}$$

where:

C_t = Concentration of chemical in wet tissue (either whole fish or specified tissue)
f = Fraction lipid content in the organism

The lipid fraction of the edible muscle tissue is generally estimated because this is where the lipophilic chemicals will reside. However, the lipid content of muscle tissue can vary considerably among freshwater sport fish species (see Table I-1) as well as among the same species of different sizes and in different habitats. For this document, the rainbow trout lipid muscle content (4%) is used as the basis for point estimate BAFs for lipophilic organic chemicals. The rainbow trout is a common freshwater sport fish species caught and consumed in California and represents a reasonable "average' lipid content value among California sport fish. However, muscle lipid content can increase well above 10% in some fish species (carp, lake trout, and certain catfish) as they reach maximum size and age. The BAFs determined in this document may underestimate chemical intake if proportionally high consumption rates of such fish occur.

The tendency of an organic compound to bioconcentrate has been shown to be related to its lipophilicity and inversely related to the chemical's water solubility. However, correlations between bioconcentration and physical properties are poor for very large molecules of high molecular weight and for chemicals metabolized by fish (Oliver and Niimi, 1985). Large molecules (about 300 to 500 MW) appear to be less efficiently transferred from water and food to fish tissues, but can have very long half lives in lentic/lotic environments (U.S. EPA, 2003a). Comparison of laboratory and field bioaccumulation studies in fish show that use of laboratory BCFs (kinetic and steady state studies), in which water was the only media for bioconcentration, would severely underestimate the field residue levels of large organic molecules in fish, particularly if they are poor substrates for metabolic enzymes. This is a clear indication that water is not the primary route of fish exposure for these chemicals; consumption of contaminated food is likely the major chemical source.

U.S. EPA (1998) derived some BAFs from field measured biota-sediment accumulation factors (BSAFs) for very hydrophobic, organic compounds such as PCDD/Fs. The BSAF is the ratio of the lipid-normalized concentration of a chemical in tissue to its organic carbon-normalized concentration in surface sediment. Water concentrations of highly hydrophobic compounds can be difficult to measure accurately for field-measured BAFs, so U.S. EPA (2003a) recommends the BSAF as the only field-based method that can be used to estimate the concentration of certain organic compounds in ambient

water. The California "Hot Spots" PCDD/F BAF point estimates discussed below in Section I.3.1.6 were derived from field-measured BSAF data by U.S. EPA (1998).

U.S. EPA (1998) recommends that for organic chemicals with a log K_{ow} greater than four, the concentrations of particulate organic carbon (POC) and dissolved organic carbon (DOC) in the ambient water should be either measured or reliably estimated. For these chemicals, the concentration of the chemical that is dissolved in ambient water excludes the portion sorbed onto particulate or dissolved organic carbon. The freely dissolved concentration is considered to represent the most bioavailable form of an organic chemical in water and, thus, is the form that best predicts bioaccumulation. The freely dissolved concentration is calculated as:

$$C^{fdw} = (f_{fd}) \times (C^{tw}) \qquad \text{Eq. I.3}$$

Where:
C^{fdw} = freely dissolved concentration of the organic chemical in ambient water
f_{fd} = faction of the total chemical in ambient water that is freely dissolved
C^{tw} = total concentration of the organic chemical in ambient water

If F_{fd} is not known, it may be calculated using the equation:

$$F_{fd} = \frac{1}{1 + POC \times K_{ow} + DOC \times 0.08 \times K_{ow}} \qquad \text{Eq. I.4}$$

For the California BAFs, DOC and POC were sometimes based on U.S. EPA (2003a) national default estimates of 2.9 mg/L for DOC and 0.5 mg/L for POC. These values reflect the central tendency estimated for DOC and POC for lakes and reservoirs distributed throughout the United States.

Field-based estimates of the freely dissolved concentration of an organic chemical in water (C^{fdw}) are preferred in order to predict BAF point estimates. However, Eq. I.4 was used to estimate f_{fd} in a number of instances when sufficient data were lacking in studies used to estimate a BAF.

I.1.2 Uptake and Accumulation of Inorganic and Organic Metals in Fish Tissues

In aquatic systems the availability of a metal to fish depends on many physico-chemical as well as biological factors. As summarized by Dallinger et al. (1987), availability is influenced by the chemical speciation of the ionic forms. The chemistry of the water including factors such as pH, hardness, and the presence of organic compounds and suspended particles may change the activity of free metal ions and influence the speciation of heavy metals. Binding to, and release from the sediment also affects the availability of metals to fish. Among the biological factors affecting metal availability, species-specific differences like feeding behavior and habitat preferences play a dominant role. These basic features are modified by physiological factors, such as

accumulation rates and the binding capacity in various fish species. The three ways by which inorganic metals may enter fish include body surface, the gills, and the alimentary tract. However, fish seem to be able to homeostatically regulate some heavy metals that they are exposed to. Thus, BCFs and BAFs for metals will generally be smaller compared to BCFs and BAFs for persistent bioaccumulative organic chemicals.

In general, soluble metal fractions may accumulate preferentially via the gills, and particulate metal fractions via the alimentary tract (Dallinger et al., 1987). Unlike persistent, hydrophobic organic chemicals, bioconcentration and biotransferance factors of metals tend to decrease with increasing trophic level up to fish, although the organometal methylmercury is an exception. However, even if biomagnification is not observed, or bioconcentration factors are small, the amount of metal transferred via food or water can be high enough to reach levels that are harmful to humans. This is because under chronic exposure of a water system, very high metal levels may occur in sediments, macrophytes and benthic animals in relation to the water levels. Thus, ingestion of sediment and sediment-dwelling invertebrates by bottom-dwelling fish species may be an important route of metal uptake by these fishes.

The wet weight muscle tissue concentrations of metals are used for determination of the BAF values. If the reference data are expressed only as a dry weight muscle tissue concentration, the tissue concentration was adjusted to a wet weight concentration using a factor of 0.24 (i.e., water content of fish muscle is roughly 75-76% by weight) if specific conversion data are not presented in the reference to calculate the adjustment.

An inverse relationship between metal accumulation and weight/size of the fish has been observed; metal in tissues decreases with increasing size or weight of fish (Liao et al., 2003). This effect has been attributed to growth dilution, increased metabolic rate in juvenile fish and increased ability to depurinate the metals as the fish matures. As a result, metal uptake studies in fingerlings or juvenile fish may overestimate bioaccumulation of mature sport fish caught and consumed by anglers and were usually not used in this document to derive accumulation factors.

Another factor to take into account is exposure duration. Numerous accumulation studies summarized below have observed long exposure times, on the order of months, before steady-state levels of a metal are reached in fish tissues. Thus, short-term exposure studies may underestimate bioaccumulation of a metal in fish.

Based on the bioaccumulation literature for metals of interest in the “Hot Spots” program, some general statements can be made. Waterborne exposure to an inorganic metal will result in greatest metal accumulation in gill, kidney and liver. Metals in the diet will increase levels in the gut as well. Muscle tissue will have the lowest accumulation of the metals. Basing BAFs on whole body concentrations of a metal may overestimate metal intake, as the concentration of an inorganic metal can be quite high in the viscera (e.g., kidney and liver), with organ-specific BAFs of 1000 or greater. Where sufficient data were present, laboratory-measured BCFs were lower for a metal than those derived using data from field studies. BCF studies often did not account for

intake via contaminated food, which in some studies summarized below was shown to be an important route of exposure for inorganic metals. Also, many of the laboratory BCF studies likely did not attain steady-state concentrations because exposures were too short.

In almost all instances, acidic water bodies (generally with a pH of 6.5 or lower) will increase accumulation of the cationic metals and oxy-anionic chromium in fish organs and tissues compared to pH neutral (7.0 to 7.5) water bodies. The default BAFs in this document are primarily based on pH neutral lentic water bodies, as these are the most common in California. Consequently, the default BAFs may underestimate the actual accumulation of a metal in fish if the water body is acidic.

I.2 Derivation of Fish BAFs

I.2.1 Semi- or Non-Volatile Organic Chemicals

I.2.1.1 Diethylhexylphthalate (DEHP)

DEHP has been detected in marine and lake sediments, as well as in marine and freshwater sport fish (Stalling et al., 1973; McFall et al., 1985; Camanzo et al., 1987; Mackintosh et al., 2004). However, the source of the DEHP found in these marine and lake sediments is not likely to be solely from air emissions. The very high K_{ow} of 7.73 and model calculations suggest that DEHP could readily bioaccumulate in fish and that dietary uptake would be an important route of exposure (Staples et al., 1997; Gobas et al., 2003). However, bioaccumulation and biomagnification studies of DEHP in fish show roughly three orders of magnitude lower BCFs/BAFs than predicted based on the K_{ow} of DEHP. This finding is a result of trophic dilution and lack of biomagnification through the aquatic food web, primarily due to the metabolic transformation of DEHP in fish (Staples et al., 1997; Mackintosh et al., 2004). The term trophic dilution means that the BAF tends to decrease as the trophic level increases.

The only freshwater study from which a field-measured BAF was developed was based on a Dutch study investigating the occurrence of DEHP in the freshwater and fish throughout the Netherlands (Peijnenburg and Struijs, 2006). Twenty-five samples of bream and roach fish and 66 freshwater samples from 23 sites were collected throughout the country. Based on the geometric mean DEHP concentration of 1.8 µg/kg wet fish and the dissolved freshwater DEHP concentration of 0.33 µg/L, a BAF of 5.5 is calculated (Table I.3). We corrected for the lipid fraction in the whole fish samples (median: 0.5% lipid), generating a lipid-normalized DEHP BAF of 1.1×10^3. Finally, we also corrected for the muscle lipid content of rainbow trout (4%), which is approximately eight times greater than that of the bream and roach fish, generating a BAF of 44.

An assumption used for this BAF is that the influence of collecting fish and water samples at different times and from different locations on this BCF is not large. Another factor to consider is that the fish in the Dutch study were collected from both lentic and lotic water bodies. Lentic environments are characterized by still (not flowing) water, as

in lakes and reservoirs. But the lotic environments are characterized by flowing water, as in streams and rivers.

Gobas et al. (2003) and Mackintosh et al. (2004) conducted a saltwater field study to assess the food-web bioaccumulation of a range of phthalate esters including DEHP. The calculated lipid-normalized BAF for the staghorn sculpin, a forage fish, and the dogfish, a predatory species, were 16,000 and 580, respectively (Table I.3). The larger dogfish (3 kg BW) has a smaller BAF than the sculpin (0.1 kg BW) due to gill elimination and fecal egestion rates dropping with increasing organism size and becoming negligible compared to growth rates.

Table I.3. BAF Values for DEHP in Fish

Fish Species	Total BAF[a]	BAF(fd)[b]	BAF(rt)[c]
Staghorn Sculpin	ND[d]	16,000	640
Spiny Dogfish	ND	580	23
Bream & Roach	5.5	1091	44

[a] Total concentration in whole fish divided by the total concentration of chemical in water
[b] Freely dissolved, lipid-normalized concentration
[c] BAF(rt) for sport-sized rainbow trout (rt) based on muscle lipid content of 4%
[d] No data

Supporting studies from other laboratories report BCFs in small sport and non-sport fish. Whole-fish BCFs of 17 and 30 were estimated in separate studies in small rainbow trout (Mehrle and Mayer, 1976; Tarr et al., 1990). Mayer (1976) estimated a BCF of 594 in fathead minnows, and Karara and Hayton (1984) estimated a BCF of 637 in sheepshead minnows. The estimated BCF values are based on the parent compound (i.e., they did not estimate a total BCF including DEHP and its metabolites) and did not include data that appeared to suffer from water solubility problems or lack of steady state attainment.

Basing the bioaccumulation of DEHP on BCF values does not take into account accumulation of DEHP from food or sediment sources, which may result in an underestimation of the BAF. In addition, basing a BAF on fingerlings or small fish may overestimate BAFs for sport-sized fish. Until field-based bioaccumulation studies for specific lentic water bodies are published for DEHP, we recommend that the BAF of 44, based on the Dutch freshwater field study, be used in the “Hot Spots” program as the default point estimate for DEHP accumulation in sport fish.

I.2.1.2 Hexachlorobenzene

HCB in the atmosphere is predicted to be predominantly in the vapor phase (see Appendix E). HCB concentrations in the vapor phase averaged 96.6% (range: 92-100%) of the total HCB concentration in air samples over Ontario, Canada (Lane et al., 1992). This finding would suggest that airborne deposition of HCB into water bodies would be small enough to disregard. However, due to the extreme persistence of HCB in air, water and soil, accumulation of HCB into water bodies by both dry and wet

deposition can be significant (Eisenreich et al., 1981; Kelly et al., 1991). Field studies at Lake Superior, a relatively pristine water body in which organics deposit primarily from atmospheric sources, report HCB in water, sediment and fish tissue samples (Eisenreich et al., 1981).

Niimi and Oliver (1989) determined the percent lipid content and HCB concentration in muscle tissue of four salmonid species (brown, lake, and rainbow trout and coho salmon) collected from Lake Ontario. Based on the published water concentration of HCB in Lake Ontario, the researchers calculate a total BAF of 101,333. The total BAF was lipid-normalized based on 4% muscle lipid content in the fish, and adjusted for the concentration of freely dissolved HCB in water, assuming a DOC content of 0.25 mg/L in Lake Ontario from Gobas (1993). The resulting BAF(fd) is 2.6×10^6.

We did not adjust the BAF(fd) to the muscle lipid fraction of rainbow trout (0.04) used in the California "Hot Spots" program because it is the same as the fish investigated by Niimi and Oliver (1989). We calculated the freely dissolved HCB fraction in water (0.78) from Eq. H.4 using the national default DOC and POC content of lakes and reservoirs (U.S. EPA, 2003a). A final BAF point estimate of 81,120 ($2.6 \times 10^6 \times 0.04 \times 0.78$) is recommended for California fish.

U.S. EPA (1998) calculates a similar BAF(fd) of log 6.40 (2.5×10^6) using Lake Ontario whole fish HCB data from Oliver and Niimi (1988). This BAF(fd) is similar to that estimated by Niimi and Oliver (1989) using only the muscle HCB concentration (BAF(fd) = 2.6×10^6) of the fish presented. U.S. EPA (1998) also calculated a mean log BAF(fd) of 5.70 (5.0×10^5) derived from BSAF data for HCB. Pereria et al. (1988) and Burkhard et al. (1997) determined a similar log BAF(fd) in the range of 6.03 to 6.68 for bioaccumulation of HCB in small, mostly non-sport fish in estuarine environments.

I.2.1.3 Hexachlorocylcohexanes

Technical grade hexachlorocyclohexane (HCH) generally consists of five isomers, including α-, β-, γ-, δ-, and ε-HCH. α-HCH is the most common isomer in technical grade HCH, and γ-HCH, also known as lindane, is most often isolated and used for its insecticidal action. Consequently, most environmental fate and bioaccumulation studies have investigated the α- and γ-isomers.

Lindane is a relatively small MW compound with a short half-life in fish, so rapid equilibrium occurs between the chemical concentration in fish and the water (Oliver and Niimi, 1985). The short half-life is probably a result of its log $K_{ow} < 4$. The high chlorine content of HCHs prevents metabolism of the isomers by rainbow trout (Konwick et al., 2006). The half-life of lindane in sport-sized fish (11-13 days) is longer than in juvenile fish (about 4 days). However, Geyer et al. (1997) report that α-HCH has a longer half-life of 14.8 days in juvenile rainbow trout. In addition, they observed a positive correlation for fish lipid content and the BCF for lindane.

The major factor governing residue levels for HCHs appears to be the chemical concentration in the water (Oliver and Niimi, 1985). Thus, good agreement between field BAFs and laboratory BCFs in rainbow trout is achieved. For lindane, the whole-fish laboratory BCF was 1200 and the whole-fish field BAF in Lake Ontario fish was 1000. For α-HCH, the whole-fish laboratory BCF was 1600 and the whole-fish BAF in Lake Ontario fish was 700.

In a subsequent comprehensive investigation at Lake Ontario, Oliver and Niimi (1988) report total BAFs for α-HCH and lindane of 5357 and 9333, respectively. The lipid-normalized whole fish BAFs shown in Table I.4 were based on a weighted average lipid content of 11% for the four fish species examined (i.e., brown, lake, and rainbow trout, coho salmon).

Normalizing the BAFs to represent the freely dissolved fraction in water based on the national default DOC and POC values for lakes and reservoirs had little effect on the freely dissolved fraction of the HCHs, as chemicals with log Kow < 4 (the lindane and α-HCH log K_{ow}s are 3.67 and 3.78, respectively) will not partition significantly to OC. Normalizing the muscle concentration of the HCHs based on the muscle lipid content of rainbow trout (4%) results in point estimate BAFs of 3394 for lindane, and 1948 for α-HCH.

Table I.4. BAF Values Based on Lake Ontario Salmonids

HCH Isomer	Total BAF[a]	BAF(fd)[b]	BAF(rt)[c]
Lindane (γ-HCH)	9333	84,845	3394
α-HCH	5357	48,700	1948

[a] Total concentration in whole fish divided by the total concentration of chemical in water
[b] Freely dissolved, lipid-normalized concentration based on 11% lipid content in whole fish
[c] BAF point estimates based on muscle lipid content of 4% for sport-sized rainbow trout

Niimi and Oliver (1989) determined the percent lipid content and HCH concentrations in muscle tissue, rather than only whole fish (apparently from the same fish examined in their previous study). The HCH concentrations in muscle adjusted for an average muscle lipid content of 4% for rainbow trout are 5.7 and 1.4 µg/kg for α-HCH and lindane, respectively. Using the water concentrations of 2.8 and 0.3 ng/L for α-HCH and lindane, respectively, from Oliver and Niimi (1988) provides BAFs of 2036 (α-HCH) and 4667 (lindane).

Because the muscle HCH concentration data in Niimi and Oliver (1989) was at or below the limit of detection for some fish, particularly for lindane, the California BAF point estimate is based on the Oliver and Niimi (1988) data presented in Table I.4. We recommend a BAF(rt) point estimate of 2671 for the “Hot Spots” program, which is the arithmetic average of the muscle tissue BAF(rt)s for the two major HCH isomers in Table I.4.

I.2.1.4 Polycyclic Aromatic Hydrocarbons (PAHs)

Polycyclic aromatic hydrocarbons (PAHs) are compounds with two or more fused benzene rings and often contain alkyl side groups. In water and sediment, low molecular weight PAHs (i.e., containing two or three aromatic rings) are more easily degraded by microbes, whereas the high molecular weight PAHs (i.e., containing four or more aromatic rings), including benzo[a]pyrene (BaP), tend to persist (Meador et al., 1995).

Bioaccumulation of PAHs in fish has not been rigorously studied, in part because PAHs undergo liver metabolism in fish resulting in low to non-detectable concentrations of the parent PAHs in fish tissues (Meador et al., 1995). Bioaccumulation of PAHs tends to decline with increasing K_{ow}, probably due to low gut assimilation efficiency and increased metabolism. However, low molecular weight PAHs tend to be less persistent in fish than the high molecular weight PAHs, probably due to more ready diffusion in and out of lipid pools.

BaP has been shown to be extensively metabolized in fish. In small bluegill sunfish (4 to 12 g wet weight) exposed to ^{14}C-labelled BaP in water, only 5% of the radiolabel in whole fish samples at the end of 24 hr exposure was found to be the parent compound (McCarthy and Jimenez, 1985). In their risk assessment, Boyce and Garry (2003) estimated a whole fish BCF of 14 for BaP based on the average value reported from relevant laboratory bioaccumulation studies in the literature.

Using the assumption that a typical lipid fraction of whole fish is 0.05 (Staples et al., 1997), and a muscle/whole body lipid ratio of 0.20 for adult rainbow trout (Niimi and Oliver, 1983), we calculated the lipid-normalized muscle tissue BCF as 56 for BaP. Adequate data for the DOC and POC water concentrations were not supplied by the studies used to derive the BCF, so the influence of this factor on the BAF could not be accounted for in the final estimate.

Burkhard and Lukasewycz (2000) determined field-measured BAFs for several PAHs found in water, sediment and lake trout muscle lipid of Lake Superior. The total BAF and BAF(fd) in Table I.5 were calculated by the researchers for lake trout in Lake Superior. The BAF(rt) was calculated by OEHHA for PAHs in rainbow trout (4% muscle lipid content) using default DOC + POC content for U.S. lakes and reservoirs. The relative order of metabolism was obtained by dividing the BAF of the chemical by its corresponding K_{ow}. By increasing rate of metabolism in the fish, the relative order was pyrene, benz[*a*]anthracene, chrysene/triphenylene, fluoranthrene, and phenanthrene. Thus, metabolism of the parent PAH compound appears to primarily control accumulation in the muscle tissue.

Table I.5. BAF Values for Polycyclic Aromatic Hydrocarbons

PAH congener (# of rings)[a]	PEF[b]	Total BAF[c]	BAF(fd)[d]	BAF(rt)[e]
Phenanthrene (3)	ND[f]	18	89	4
Fluoranthrene (4)	ND	331	1660	62
Pyrene (4)	ND	10,471	52,481	2067
Benz[*a*]anthracene (4)	0.1	9550	53,703	1573
Chrysene/triphenylene (4)	0.01 (chrysene only)	759	4074	124[g]

[a] Number of benzene rings per PAH compound shown in parentheses
[b] Potency Equivalency Factor for carcinogenicity, using benzo[a]pyrene as the index PAH compound with a PEF=1.
[c] Total concentration in fillet of lake trout divided by the total concentration of chemical in water
[d] Freely dissolved, lipid-normalized concentration based on 20.5% lipid content in fish fillet samples
[e] BAF point estimates based on muscle lipid content of 4% for rainbow trout and default DOC + POC content for U.S. lakes and reservoirs from U.S. EPA (2003a).
[f] Not determined, as a result of inadequate or no evidence for carcinogenicity in animals.
[g] Assumed to represent BAF(rt) for both chrysene and triphenylene

The data in Table I.5 suggest that PAHs with four rings are more likely to accumulate in fish than PAHs with three rings. A study by Zabik et al. (1996) found some five- and six-ring PAHs in muscle fat of lake trout from Lake Superior. This study did not detect BaP in the fish tissue, but did find dibenzo[ah]pyrene which has a potency equivalency factor (PEF) value of 10. BAFs could not be calculated for any PAHs with five or more rings, either because dissolved levels of these congeners could not be detected in the water, or because the congener could not be detected in the fish (Baker and Eisenreich, 1989; 1990; Zabik et al., 1996). Another reason is that the individual PAHs quantified in water and fish were not all the same between various studies.

We calculated an average BAF(rt) of 849 from the congener groups in Table I.5 that have PEFs (i.e., benz[*a*]anthracene and chrysene), and is recommended as the default point estimate of BAF(rt) for PAHs. Considering that measurable levels of high molecular weight carcinogenic PAHs have been detected in fish muscle (although not enough data are present to estimate BAFs), but that a BAF for BaP is likely below the BAF(rt) of 849, a point estimate based on the most bioaccumulative carcinogenic PAHs should be sufficiently health protective to avoid underestimation of a BAF for the carcinogenic PAHs.

I.2.1.5 Polychlorinated biphenyls (PCBs)

PCBs are a group (209 congeners) of organic chemicals, based on various substitutions of chlorine atoms on a basic biphenyl molecule. However, probably less than 100 congeners are found at concentrations of significance in commercial PCB mixtures and environmental samples, and fewer represent a toxicological concern (Niimi, 1996). Solubilities and octanol-water partition coefficients (K_{ow}) for PCB congeners range over several orders of magnitude. The K_{ow}s, which are often used as estimators of the potential for bioconcentration, are highest for the most chlorinated PCB congeners.

Since log K_{ow} values of most PCB congeners are higher than 5, biomagnifications through trophic transfer is the primary mechanism governing the accumulation of these compounds in fish (Oliver and Niimi, 1985; van der Oost et al., 2003). Thomann and Connolly (1984) demonstrated that more than 99% of PCBs in Lake Michigan lake trout came from food. A food web bioaccumulation PCB study by Morrison et al. (1997) noted that over 99% of PCB 153 accumulated in fish through consumption of contaminated food and 79.9% of PCB 42 accumulation was through food (PCB 42 has a lower K_{ow}).

Food-web relationships and biomagnification may be more related to the PCBs in sediment rather than water. Therefore, biota sediment accumulation factors (BSAF) have been developed for PCBs as an indicator of bioavailability to fish because sediment is an important source for hydrophobic chemicals such as PCBs (Niimi, 1996). However, the PCBs found in the highest concentrations in fish generally reflected their high concentrations in water and sediment (Oliver and Niimi, 1988).

In the comprehensive field study by Oliver and Niimi (1988), the most common classes of PCB isomers in various salmon and trout species from Lake Ontario were the penta- and hexachlorobiphenyls, making up about 65% of the total isomeric composition. The tetra- and heptachlorobiphenyls made up another 30% of the isomeric composition. Eleven single and co-eluting PCB congeners (153, 101, 84, 138, 110, 118, 180, 87 + 97, 149, 187 + 182, and 105) constituted over half the PCBs in fish. The single most common congener was 153 (2,2', 4,4',5,5'-hexachlorobiphenyl). The tri, tetra, and penta congeners comprised a much higher fraction in water than in the fish. Thus, the PCB accumulation pattern in fish is not an accurate reflection of the aqueous composition of the mixture found in the lake.

Because the calculated total BAFs for the most common PCBs accumulating in fish gave a roughly 10-fold range for the values, a weighted average total BAF was calculated for the four most common chlorinated classes of PCB congeners in fish from the study by Oliver and Niimi (1988). These were the tetra-, penta-, hexa-, and hepta-CBs, which constituted about 95% of the overall PCBs accumulated in whole fish. The resulting weighted-average total BAF was 6.12×10^6.

We calculated a lipid-normalized BAF of 5.56×10^7 based on the whole fish lipid content of 11% determined in the study by Oliver and Niimi (1988). The mean percent contribution of PCB congeners was similar for whole fish and muscle among the species even though total concentrations vary widely (Niimi and Oliver, 1989). Consistency among congener contribution in whole fish and muscle was also demonstrated by cumulative percent of the more common PCB congeners. The freely dissolved PCB portion in water is based on data by Gobas (1993) who found about half of total PCBs in Lake Ontario water was in the freely dissolved form. The resulting calculated lipid-normalized, freely dissolved BAF, or BAF(fd), is 1.11×10^8.

Next, we adjusted the BAF(fd) to generate a BAF point estimate to be used in the California "Hot Spots" program. Correcting the BAF(fd) for the muscle lipid fraction of 0.04 in rainbow trout, and correcting for the freely dissolved PCB fraction in water (0.25, or 50% of that calculated for Lake Ontario) gives a final BAF point estimate of 2.22×10^6 ($1.11 \times 10^8 \times 0.04 \times 0.50$).

I.2.1.6 Polychlorinated Dibenzo-p-Dioxins and Dibenzofurans (PCDDs and PCDFs)

PCDDs and PCDFs are two groups of toxic compounds composed of 135 and 75 individual isomers, respectively. Most studies have focused on the 17 congeners with lateral Cl substitutions at the 2,3,7,8 positions (Niimi, 1996). These congeners appear to be primarily responsible for the accumulation and toxicity of PCDD/Fs. The 2,3,7,8-TCDD, 1,2,3,7,8-PCDD, 2,3,7,8-PCDF and 2,3,4,7,8-PCDF congeners were common in four fish species (brown trout, lake trout, rainbow trout, coho salmon) examined from Lake Ontario. Dietary uptake of PCDD/Fs appears to be of more importance than waterborne uptake, although dietary absorption efficiencies in fish are consistently lower and more variable compared to PCBs.

The two main lateral substituted PCDDs, 2,3,7,8-TCDD and 1,2,3,7,8-PCDD, constituted about 89% of the sum of all PCDDs in the fish (Niimi, 1996). The two main PCDFs, 2,3,7,8-PCDF and 2,3,4,7,8-PCDF, constituted 51% of the sum of all PCDFs in the fish. Since these congeners are the most bioaccumulative and have the greatest toxicity concern, the PCDD/F BAFs will be representative of these four congeners.

U.S. EPA (1998) derived lipid-normalized, freely dissolved BAFs (i.e., BAF(fd)) from field measured BSAFs. The high hydrophobic nature of PCDD/Fs makes it difficult to accurately determine field-measured BAFs (i.e., based on water concentrations) for this group of chemicals. U.S. EPA (2003a) recommends the BSAF as the only field-based method that can be used to estimate the concentration of these compounds in ambient water. Using a weighted-average approach for the main congeners found in fish, the BAF(fd)s were 1.00×10^7 and 5.50×10^6 for PCDDs and PCDFs, respectively.

We then adjusted the BAF(fd)s to generate BAF point estimates to reflect the muscle lipid fraction of rainbow trout (0.04) for the "Hot Spots" program. The final BAF point estimates of 400,000 and 220,000 were calculated for PCDDs and PCDFs, respectively, for California fish. The average BAF of these two values, 310,000, is the recommended BAF point estimate for the "Hot Spots" program.

I.2.2 Derivation of Fish BCFs – Metallic and Organometalllic Compounds

I.2.2.1 Arsenic

Inorganic arsenic (As), either as As(III) or As(V), are the predominant forms in aquatic ecosystems such as sediment and water, but organoarsenic compounds may be present at significant levels in freshwater fish. Average concentrations of As in ambient freshwater are generally <1 to 10 µg/L (U.S. EPA, 2003b). U.S. EPA (2003b) states

that recent research shows each of the major inorganic and organic As species, including As(III), As (V), arsenobetaine (AsB), dimethylarsenic acid (DMA), and monomethylarsonic acid (MMA), may exhibit different toxicities, and it may be important to take into account the fraction of total As present in the inorganic and organic forms when estimating the potential risk posed through consumption of As-contaminated fish. Ideally, the most appropriate BAFs would incorporate the most bioavailable and toxic form(s). This is currently not possible, so the point estimate BAF in this document will be based on total As in sport fish muscle tissue.

Direct accumulation of As in tilapia was proportional to the concentration of arsenicals in water (Suhendrayatna et al., 2002). Approximately 25% of absorbed arsenic from water in whole fish as either As(III) or As(V) was transformed to methylated arsenic, primarily methyl-, dimethyl-, and trimethyl- forms. Whether absorbed as As(III) or As(V) from water, metabolism in fish resulted in roughly equivalent concentrations of both inorganic arsenic species in whole fish, although As(III) was absorbed more easily than As(V).

Accumulation and transformation of As in the food chain has been investigated. In a three-step freshwater food chain (algae-shrimp-tilapia), exposure to As(III) in water resulted in total As concentrations decreasing in the organisms with each step up the food chain (Suhendrayatna et al., 2002). Inorganic As species were the predominant forms in each organism (As(III), 9-41%; As(V), 50-90%), with only a limited degree of As methylation at each step in the food chain. However, when As(V) was the dominant As species in water, mouthbreeder fish raised long-term in aquaculture ponds contained predominantly organoarsenic species in muscle tissue, with inorganic As equaling only 7.4% of total As (Huang et al., 2003).

Predicted and measured As concentrations in major organs of tilapia from culture ponds high in As observed highest As concentrations in the alimentary canal, blood and liver, and lowest concentrations in muscle tissue (Liao et al., 2005). Steady-state concentration of As in muscle tissue took up to 300 days to be achieved.

Arsenic bioaccumulation studies in fish have been conducted in laboratory, aquaculture pond, and field investigations, although exposure durations to achieve steady-state concentrations in fish tissues were only observed for the aquaculture and field studies. The BAFs findings are presented in Table I.6.

In aquaculture studies, an average BCF of 8.2 (range: 5.4 to 11) was determined for bioconcentration of As in muscle of mouthbreeder fish raised long-term in ponds from three different regions in Taiwan (Huang et al., 2003). The fish were collected from ponds containing 14.4 to 75.8 µg/L As in water. A BCF of 3.5 was recorded for As in muscle tissue of large-scale mullet raised in a Taiwanese aquaculture pond (Lin et al., 2001). In farmed tilapia fish exposed to As in water for 300 days, a muscle BCF = 4 was calculated (Liao et al., 2005). In a similar study, BCFs of 15 and 53 were obtained for As from tilapia muscle raised in two aquaculture ponds containing 49.0 and 17.8 µg/L As in water, respectively (Liao et al., 2003). Because the fish in these aquaculture

studies were fed with artificial bait that did not contain As, the accumulation factors may better represent BCF values rather than BAF values.

Only two field studies were located that presented data to determine a muscle tissue BAF for fish in As-contaminated lentic water bodies. A BAF of 28 was determined from muscle tissue of the common carp exposed to As in four wastewater treatment basins in Pennsylvania (Skinner, 1985). Channel catfish and large-mouth bass from a reservoir impacted by mining and agricultural runoff had muscle BAF values of 12.5 for As (Baker and King, 1994).

Table I.6. BAFs for Arsenic in Muscle Tissue of Fish from Lentic Water Bodies

Location	Species	Arsenic Water Concentration	Arsenic Muscle Concentration	BAF	Reference
Taiwanese Aquaculture Studies					
Putai Pond	mouthbreeder	75.8 µg/L	0.41 µg/g	5.4	Huang et. al., 2003
Yichu Pond	mouthbreeder	15.1	0.12	7.9	Huang et. al., 2003
Hsuehchia Pond	mouthbreeder	14.4	0.16	11.1	Huang et. al., 2003
Putai Pond	large-scale mullet	169.7	2.41	14.2	Lin et. al., 2001
Hsuehchia Pond	tilapia	17.8	0.95	53.4	Liao et. al., 2003
Yichu Pond	tilapia	49.0	0.75	15.3	Liao et. al., 2003
Tilapia farms	tilapia	94	1.5	16	Liao et al., 2005
Field Studies					
San Carlos Reservoir, AZ	large-mouth bass	8	0.1	12.5	Baker & King, 1994
San Carlos Reservoir, AZ	channel catfish	8	0.1	12.5	Baker & King, 1994
Wastewater treatment basins, PA	common carp	3.0 – 16.0	0.22 - <0.05	28	Skinner, 1985

Among the studies presented in Table I.6, average BCF/BAFs were calculated for six fish species: 8.1 for mouthbreeder, 14.2 for large-scale mullet, 28 for tilapia, 12.5 for large-mouth bass and channel catfish, and 28 for common carp. The arithmetic average BAF combined for all species is 17, which we recommend as the BAF point estimate for As.

I.2.2.2 Beryllium

Little information could be found for bioaccumulation of beryllium in fish. U.S. EPA (1980) estimated a BCF of 19 in whole bluegill after 28 days of exposure in water. It is unknown if steady state levels were attained in the fish, although the whole-body elimination half-life was observed to be one day. Limited data by Eisler (1974) suggest that whole-fish accumulation of inorganic beryllium in mummichogs from seawater is similar to some other cationic metals such as cadmium, in that whole fish uptake of beryllium appears to be a passive process.

No information could be found regarding the accumulation of beryllium in muscle tissue of fish. Based on BCF and BAF studies of other cationic metals discussed in this appendix, steady state levels were probably not reached in bluegills during the 28-day exposure (U.S. EPA, 1980). The muscle BAFs for other cationic metals (i.e., cadmium, inorganic mercury, lead, nickel) presented in Table H.2 range from 20 to 80. We recommend that a mean cationic metal BAF of 40 be used for beryllium in sport fish until more comprehensive bioaccumulation studies are conducted.

I.2.2.3 Cadmium

A considerable number of cadmium (Cd) bioaccumulation studies have been carried out in fish. Freshwater sport fish accumulate Cd mainly in gills, kidney, liver, and gastrointestinal tract (Sangalang and Freeman, 1979; Harrison and Klaverkamp, 1989; Spry and Wiener, 1991; Szebedinszky et al., 2001). However, Cd does not accumulate as appreciably in muscle tissue of exposed sport fish and the concentration is generally low relative to other tissues and organs.

The Cd concentration in fish varies with the proportion of free divalent Cd in water, typically increasing with increasing water concentration (Camusso et al., 1995). Direct uptake across the gills has been generally considered the primary influx of the metal for fish in dilute waters (Spry and Wiener, 1991). However, absorption of Cd from contaminated food sources can be a significant route of exposure, and may be the dominant source of Cd in bodies of water with high pH and calcium levels (Ferard et al., 1983; Harrison and Klaverkamp, 1989; Farag et al., 1994; Kraal et al., 1995; Thomann et al., 1997).

The main characteristics of lakes that enhance bioaccumulation of Cd in fish include low pH (pH ≤6), low aqueous calcium (often <2 mg/L), and low DOC (usually <3 mg/L) (Spry and Wiener, 1991). In the eastern U.S., whole-body Cd levels in bluegill fish from low pH lakes were as much as 10-fold higher compared to cadmium in bluegills from circumneutral-pH lakes. In addition, accumulation of Cd in fish is more sensitive to changes in water hardness, usually expressed in mg/L $CaCO_3$, rather than changes in DOC (Wiener and Giesy, 1979).

Steady-state equilibrium of Cd in muscle and other tissues was obtained in brook trout at about 20 weeks exposure in a three-generation exposure study by Benoit et al.

(1976). Benoit et al. (1976) also recorded a muscle BCF = 3.5 in brook trout exposed to aqueous Cd in Lake Superoir water for 70 weeks. Equilibrium of Cd in tissues was also reached at 20 weeks of exposure.

Perhaps significantly, the numerous laboratory studies that measured muscle Cd content show an inverse relationship with water hardness. In several laboratory studies, BCFs varied between 1.6 to 4.8 for Cd in muscle of rainbow trout, carp and brook trout with a water hardness between 33 and 93 mg /L $CaCO_3$ (Benoit et al., 1976; Giles, 1988; Harrison and Klaverkamp, 1989; de Conto Cinier et al., 1997). Exposure durations for these studies ranged from 3 to 17 months, and tissue and organ Cd concentrations increased with increasing exposure duration. Two other laboratory studies that recorded somewhat higher BCFs of 17-19 in muscle of rainbow and brook trout also had the lowest water hardness (19-22 mg /L $CaCO_3$) (Sangalang and Freeman, 1979; Kumada et al., 1980). The exposure duration of fish to Cd-contaminated water for both of these studies was about 3 months. Alternatively, laboratory studies exposing rainbow trout to Cd in water with considerably higher hardness (140-320 mg/L $CaCO_3$) at circumneutral-to-high pH (7.4-8.2) for up to 80 weeks recorded BCFs from 0 to 2 in muscle tissue (Roberts et al., 1979; Calamari et al., 1982; Brown et al., 1994; Szebedinszky et al., 2001).

The level of DOC in the water of the laboratory BCF studies above were not discussed, but were likely low. Low DOC levels would allow water hardness to be the main factor affecting bioaccumulation of Cd.

Although comparatively few field studies have been published that investigated Cd accumulation in muscle tissue of sport fish, the field study by Wiener and Giesy (1979) supports the assumption that water hardness (and perhaps pH) is a more important factor in controlling tissue accumulation then the DOC content. In this study, a Cd muscle BAF = 12 was determined in bluegill stocked in an acidic (pH = 4.6), highly organic pond for 511 days. Measured total organic carbon of the pond was anywhere from 15 to >30 mg/L, but the $CaCO_3$ content of the pond was very low, averaging 2.1 mg/L.

Two field studies examined the effect of acidified water in New York lakes on fish tissue levels of various heavy metals as a result of acid deposition (i.e., acid rain) (Heit et al., 1989; Stripp et al., 1990). In general, higher BAFs were recorded for Cd in muscle tissue of yellow perch and white sucker from the most acidic lentic water body, Darts Lake, compared to two other lakes, Rondaxe and Moss lakes, with higher pH values (Table I.7). All three lakes were clear-water lakes with comparable concentrations of DOC.

Table I.7. BAFs for Cadmium in Muscle Tissue of Fish from U.S. Lakes

Location	Species	Lake pH	Cd Water Concentration (µg/L)	Cd Muscle Concentration (µg/g)	BAF
Darts Lake (1)	White sucker	4.9-5.4	0.7	0.062	89
Darts Lake (1)	Yellow perch	4.9-5.4	0.7	0.048	69
Darts Lake (2)	White sucker	5.1-5.4	0.26	0.038	146
Darts Lake (2)	Yellow perch	5.1-5.4	0.26	0.028	108
Rondaxe Lake (1)	White sucker	5.8-6.7	1.1	0.024	22
Rondaxe Lake (1)	Yellow perch	5.8-6.7	1.1	0.024	22
Rondaxe Lake (2)	White sucker	5.8-6.7	0.61	0.025	41
Rondaxe Lake (2)	Yellow perch	5.8-6.7	0.61	0.038	62
Moss Lake (1)	White sucker	6.5-6.8	0.6	0.022	36
Moss Lake (1)	Yellow perch	6.5-6.8	0.6	0.034	56
Skinface Pond, SC (3)	Bluegill	4.6	0.17	0.0021	12

Sources: (1) Stripp et al., (1990); (2) Heit et al., (1989); (3) Wiener and Giesy (1979).

The few field studies examining muscle tissue levels of Cd in contaminated lakes indicate that basing a BAF on laboratory BCF studies would underestimate the accumulation potential of Cd in fish. However, it is probably not appropriate basing a BAF on data from highly acidified lakes (i.e., Darts Lake and Skinface Pond), as California generally does not have the lake acidification problem that exists in the northeastern U.S. Thus, we recommend default BAF point estimate for Cd of 40 based on fish from the variable pH (Rondaxe Lake) and circumneutral lakes (Moss Lake), which is the arithmetic average BAF combining both fish species (white sucker and yellow perch, which represent trophic level 3 and 4 fish, respectively) from these lakes.

I.2.2.4 Chromium

Hexavalent chromium (Cr(VI)) in water readily penetrates the gill membrane of fish and is the main route of uptake (Holdway, 1988). Organs and tissues that accumulate Cr(VI) include gills, spleen, kidney, gall bladder, gastrointestinal tract, opercular bone, and brain. Accumulation in muscle tissue is minor compared to these other tissues. No biomagnifications occur at higher trophic levels. Cr(VI) uptake is a passive process with resulting tissue concentrations directly proportional to exposure concentrations. Chromium bioavailability to fish increases with decreasing pH (7.8 to 6.5), resulting in increased bioaccumulation in tissues and organs (Van der Putte et al., 1981).

In a laboratory study, six-month exposure of rainbow trout to Cr(VI) as potassium dichromate ($K_2Cr_2O_7$) in water resulted in a muscle tissue BCF of 3 (Calamari et al., 1982).

A small freshwater aquatic ecosystem containing adult catfish was created in a small tank, and a single dose of potassium dichromate was added to the system (Ramoliya et al., 2007). After 21 days of exposure, a muscle tissue BCF <1 was calculated for the

catfish based on the average water concentration of Cr(VI) over the 21 days. However, the Cr(VI) content in the catfish had not reached equilibrium at the end of exposure, and was still increasing with increasing exposure duration. High levels of Cr(VI) in the intestine of the catfish suggest Cr(VI) may be absorbed via food sources.

Rainbow trout that were reared for two years in either a hatchery or river water that was contaminated with low levels of sodium dichromate had muscle tissue BCFs of 40 and 12, respectively (Buhler et al., 1977). Exposing the same fish to high concentrations of Cr(VI) (2.5 mg/L) for 22 days increased muscle levels of Cr(VI), but the resulting BCF was only 0.1-0.2.

Two field studies from South Africa determined the bioaccumulation of chromium in muscle tissue of fish. In adult African sharptooth catfish, muscle tissue BAFs of 10 and 16 were calculated for fish kept in a treated sewage maturation pond and in a reservoir, respectively, for 12 months (Van den Heever and Frey, 1996). Nussey et al. (2000) calculated an average muscle tissue BAF of 23.6 in the moggel, a cyprinid fish, collected from a different reservoir over a period of 15 months.

Based on the long-term field exposure studies, an average muscle BAF of 26 was calculated for rainbow trout in the Buhler et al. study, and an average muscle BAF of 13 was calculated for the African sharptooth catfish in the van den Heever and Frey study. Combined with the muscle tissue BAF of 23.6 in the moggel from Nussey et al. (2000), we calculate an arithmetic mean BAF of 21 and recommend this value as the BAF point estimate for Cr.

I.2.2.5 Lead

Similar to Cd, factors that may increase accumulation of cationic metals such as lead in fish include low pH (6.0-6.5 or less) in the water body, low concentrations of aqueous calcium that compete with lead for absorption through the gills, and low DOC (Varanasi and Gmur, 1978; Spry and Wiener, 1991; Lithner et al., 1995). Pb appears to have a greater tendency than Cd to associate with DOC and particulate matter in lake water, with accumulation in fish varying inversely with the concentration of dissolved organics in water (Wiener and Giesy, 1979). When Merlini and Pozzi (1977a) added a Pb salt to lake water, only 8% remained in the ionic form with the remainder presumably associating with dissolved organics.

Accumulation of Pb by fish typically increases with increasing exposure concentration in water, although Pb does not biomagnify in aquatic food chains (Spry and Wiener, 1991). Pb chiefly accumulates in the bone, scales, gill, kidney, and liver. Pb does not accumulate as appreciably in skeletal muscle tissue of fish. Primary mode of absorption has been suggested to be direct uptake of Pb in the ionic state across the gills, with lead from food sources being minor or insignificant (Merlini and Pozzi, 1977a; Spry and Wiener, 1991; Farag et al., 1994). On the other hand, another laboratory study found that lead uptake in fish via food was significant, if not more important than uptake via water (Vighi, 1981).

In a three-generation laboratory study, a BCF of 2 to 3 was estimated for Pb in muscle tissue of first and second generation brook trout (Holcombe et al., 1976). Exposure to Pb in water was for 38 and 70 weeks in first and second generation fish, respectively. The concentration of Pb in muscle had reached equilibrium at about 20 weeks of exposure.

Whole bluegill Pb concentrations have been shown to be as much as 10 times higher in bluegills from low-pH lakes (pH≤6.0) compared to bluegills from circumneutral-pH lakes (pH 6.7-7.5) (Spry and Wiener, 1991). In another study, whole-fish Pb levels in sunfish increased almost three-fold when lake water pH was decreased from 7.5 to 6.0 (Merlini and Pozzi, 1977b).

In other field studies, Pb accumulated to greater extent in muscle of white suckers and yellow perch from an acidic lake compared to more neutral lakes (Heit et al., 1989; Stripp et al., 1990) (Table I.8). With increasing lake acidity, muscle bioaccumulation of Pb became increasingly higher in bottom-dwelling, omnivorous white suckers compared to carnivorous yellow perch. Thus, contact with sediments by bottom-dwelling fish increases Pb bioaccumulation.

A considerably greater concentration of Pb was found in surface sediments (880-1005 µg/g) of the lakes compared to the water (2.0-3.0 ng/g) (Stripp et al., 1990). It was postulated that higher levels in fish tissues from acidic lakes result from increased mobilization of the cationic Pb species from sediments coupled with an increase in the cationic Pb species in the acidic water.

Table I.8. BAFs for Lead in Muscle Tissue of Fish from Lentic Ecosystems

Location	Species	Lake pH	Pb Water Concentration	Pb Muscle Concentration	BAF
Acidic water bodies					
Darts Lake (1)	White sucker	4.9-5.4	3.0 µg/L	0.13 µg/g	43
Darts Lake (1)	Yellow perch	4.9-5.4	3.0 µg/L	0.058	19
Darts Lake (2)	White sucker	4.9-5.4	1.5	0.13	87
Darts Lake (2)	Yellow perch	4.9-5.4	1.5	0.055	37
Acidic lakes & ponds, NJ (3)	Yellow perch	3.7-4.6	0.8 – 3.6	0.067 – 0.11	40
Variable and circumneutral water bodies					
Rondaxe Lake (1)	White sucker	5.8-6.7	2.0	0.048	24
Rondaxe Lake (1)	Yellow perch	5.8-6.7	2.0	0.058	29
Rondaxe Lake (2)	White sucker	5.8-6.7	2.3	0.050	22
Rondaxe Lake (2)	Yellow perch	5.8-6.7	2.3	0.050	22
Moss Lake (1)	White sucker	6.5-6.8	2.5	0.031	12
Moss Lake (1)	Yellow perch	6.5-6.8	2.5	0.024	10
Witbank Dam, South Africa (4)	Moggel	ND*	140	2.00	14

Sources: (1) Stripp et al. (1990), (2) Heit et al. (1989), (3) Sprenger et. al. (1988), (4) Nussey et al. (2000)
* No data

The field data indicate higher muscle BAFs in fish from highly acidified lakes (Table I.8). California generally does not have the acidification problem that exists in the northeastern U.S. Thus, a BAF point estimate for Pb was based on fish from the variable pH and circumneutral lakes. The BAF data from Nussey et al. (2000) was also included, although water pH data were not provided in the report. We calculate an arithmetic average BAF of 19 combining all fish species (white sucker, yellow perch and moggel) from these lakes and recommend this value as the Pb BAF point estimate.

I.2.2.6 Mercury (inorganic) and Methylmercury

Mercury, like other metals deposited into water, can occur in a number of physical and chemical forms. Physically, mercury can be freely dissolved or bound to organic matter or particles suspended in water. Mercury can be found as elemental mercury (Hg^0), inorganic ionic mercury (primarily Hg^{++}), or organic mercury (e.g., methylmercury (MeHg) or dimethylmercury).

Mercury (Hg) enters aquatic ecosystems primarily as inorganic Hg, but MeHg is the dominant form of Hg found in muscle tissue of freshwater fish (Spry and Wiener, 1991). MeHg has been shown to constitute virtually all, about 99% or greater, of the total Hg in

muscle of trophic level 3-4 freshwater sport fish even though much of the Hg analyzed in the water was in inorganic Hg (Bloom, 1992; Kuwabara et al., 2007). In whole fish, the proportion of inorganic Hg is greater (5% or more of total Hg) because whole body samples include visceral tissue, such as kidney and liver, which is the principal site of inorganic Hg accumulation in fish (Hill et al., 1996; Watras et al., 1998).

As summarized by Southworth et al. (2004), MeHg is produced in aquatic environments by the action of microorganisms on inorganic Hg. It can also be removed from the aquatic systems by microorganisms that demethylate MeHg. Once formed, MeHg is taken up by microorganisms, primary producers, aquatic invertebrates, and fish. MeHg in the organisms shows the classical biomagnification process, with MeHg concentration increasing with trophic level. The concentrations of MeHg that are accumulated in fish are greatly affected by the nature of the aquatic food chain, and are sensitive to factors such as aquatic community composition and productivity. In many waters, minute concentrations (<10 ng/L) of waterborne inorganic Hg are capable of sustaining MeHg production at rates high enough to support bioaccumulation of MeHg in fish to levels warranting fish consumption advisories. The concentrations of MeHg and inorganic Hg are positively related in natural waters, which would appear to support expressing a BAF for MeHg in fish as a ratio based on total or dissolved inorganic Hg in water. Calculating MeHg bioaccumulation in fish using such a ratio would be ideal for the "Hot Spots" program (i.e., estimate the concentration of dissolved MeHg in water based in the total Hg concentration deposited in water), but introduces another level of uncertainty compared to development of BAFs directly from published reports.

Using the dissolved MeHg fraction in water to derive BAFs is recommended, as this is the primary form of MeHg that is bioaccumulated in fish. MeHg is also more toxic than other forms of mercury. However, dissolved MeHg was not always the form measured in the studies U.S. EPA (2001) identified for inclusion in their database. Thus, translators were necessary to convert between other forms of Hg measured in water and dissolved MeHg for BAF calculations. For lentic systems (i.e., lakes, reservoirs and ponds), the translators that may be use in the Hot Spots program include dissolved MeHg ($MeHg_d$) over the total Hg (Hg_t) and the $MeHg_d$ over the total MeHg ($MeHg_t$). The lentic U.S. EPA translators are $MeHg_d / Hg_t = 0.032$ and $MeHg_d / MeHg_t = 0.61$.

U.S. EPA (2001) derived the mean dissolved MeHg/total Hg translator of 3.2% for lentic ecosystems, and used it to convert between other forms of Hg measured in water and dissolved MeHg for BAF calculations. Thus it can be interpreted that 3.2% of inorganic Hg that has deposited into a lake will be converted by microorganisms and found in the form of dissolved MeHg.

Table I.9 presents various BAFs for methylmercury from U.S. EPA (2001) and California data (OEHHA, 2006). Although U.S. EPA presents the geometric means of BAFs, OEHHA recommends the use of arithmetic means of the BAFs to provide a more health protective estimate. In developing their BAFs, U.S. EPA assumed that 100 percent of the mercury measured as total mercury in both trophic levels 3 and 4 was MeHg. This assumption provides a more health protective estimate.

Table I.9. Methylmercury BAFs for Lentic/Lotic[a] Ecosystems from U.S. EPA and California Data

Agency	Environment/Comments	Mean	Trophic Level	
			3	4
U.S. EPA	Lentic Only	Geometric	1.1×10^6	5.7×10^6
U.S. EPA	Lentic Only	Arithmetic	1.5×10^6	6.2×10^6
California	Lentic Alternative	Geometric	NP	NP
California	Lentic Alternative	Arithmetic	NP	NP
U.S. EPA	Lotic Only	Geometric	5.7×10^5	1.2×10^6
U.S. EPA	Lotic Only	Arithmetic	1.3×10^6	3.9×10^6
California	Lotic Alternative	Geometric	6.8×10^5	1.1×10^6
California	Lotic Alternative	Arithmetic	1.4×10^6	3.5×10^6
U.S. EPA	Lentic/Lotic Combined	Arithmetic	1.4×10^6	5.0×10^6

[a] Lentic environments are characterized by still (not flowing) water, as in lakes and reservoirs. Lotic environments are characterized by flowing water, as in streams and rivers.

In California, using a MeHg BAF developed by U.S. EPA is complicated by the large number of Hg point sources originating from legacy mining activities, a situation somewhat unique to California. Atmospheric deposition of Hg into water bodies may be overshadowed by the existing Hg already present due to legacy mining. In addition, very little published data exist for California lentic ecosystems in order to determine if total Hg concentrations are good predictors of MeHg concentration. The BAFs and translators developed by U.S. EPA were based primarily on atmospheric deposition of Hg into water bodies. Hg speciation in water and fish may be quite different depending on whether the Hg originated from mining or atmospheric deposition.

Nevertheless, OEHHA (2006) found that the national values predicted California fish MeHg concentrations very well except for some water bodies where Hg concentrations in water were statistically higher. Hg concentrations (≥0.2 ng/L) in these water bodies were found to be more than one standard derivation from the mean for other data used in these tests. We concluded that the national default values for BAFs and translators may not work well for all water bodies in California. However, based on the limited comparisons possible, BAFs and translators based on the California data and international studies (U.S. EPA database) were found to be similar.

In partial support, Kelly et al. (1995) observed that total Hg concentration was not a good predictor of MeHg concentration in stream water or in lakes in general, but it appeared to be a good predictor for lakes within individual geographic areas. In lotic ecosystems, Southworth et al. (2004) concluded that it is not valid to assume that the fraction of total waterborne Hg comprised by MeHg would remain constant while total Hg varies at high total Hg concentrations (roughly >50 ng/L) typical of systems affected by point-source or legacy contamination. However, at total Hg concentrations less than 10 ng/L, the %MeHg varies little. They postulated that such a relationship results from saturation of the ecosystems capacity to methylate inorganic Hg at high total Hg concentrations.

Although OEHHA does not currently have an oral chronic Reference Exposure Level for methyl mercury, OEHHA recommends using a translator of 3.2% to convert total Hg deposited in water to dissolved MeHg in water under the “Hot Spots” program. Additionally, the MeHg BAF = 6,200,000 (log 6.79) from Table I.9 is recommended for California sport fish caught and consumed from lentic ecosystems.

Inorganic Hg is absorbed by fish less efficiently then MeHg from both food and water, but if absorbed, is eliminated more rapidly. For example, rainbow trout fed inorganic Hg-contaminated prey resulted in Hg predominantly accumulating in the intestines, and the Hg was not significantly absorbed into the body (Boudou and Ribeyre, 1985). During the decontamination phase, Hg that had accumulated in the intestines was rapidly excreted.

In water, the most important route for uptake of inorganic Hg in fish is likely the gills, with accumulation of Hg mainly in the gills, kidney and liver (Allen et al., 1988; Gottofrey and Tjalve, 1991). Whole-body accumulation of inorganic Hg in rainbow trout and carp increases with decreasing water pH from 9 to 5, but did not reach equilibrium during a 17-day exposure in water (Wakabayashi et al., 1987).

MeHg is the primary concern for estimating Hg bioaccumulation. Since relatively little of the Hg in fish muscle is in the inorganic form, there are very little field data to estimate a BAF for inorganic Hg.

In a laboratory tank study investigating the relationship between inorganic Hg body burden levels and toxicity, a mean muscle BCF of 84 was calculated in rainbow trout exposed to HgCl in water for 60 to 130 days (Niimi and Kissoon, 1994). Steady-state levels in muscle tissue were reached by 60 days of exposure to high levels of HgCl (64 µg/L); these levels were eventually lethal to the fish. Since most lakes of concern contain inorganic Hg levels in the ng/L to low µg/L range, such high exposure conditions may not reflect an ideal situation for estimating an inorganic Hg BAF. In addition, it has been found that food sources containing inorganic Hg are also important for fish Hg bioaccumulation (Hill et al., 1996).

U.S. EPA (2001) has used a national criteria of 51 ng/L of total Hg in water as a measure that may result in the MeHg concentration of concern of 0.3 µg/g in fish. Using the assumption that, at most, 1% of the MeHg concentration in fish muscle is actually inorganic Hg, a BAF of 59 for inorganic Hg is calculated (0.3 µg/g (0.01) ÷ 51 ng/L). Although this BAF derivation is a rather crude estimate of the inorganic Hg BAF, the value is near that calculated from the BCF study (BCF = 84) by Niimi and Kissoon (1994). OEHHA recommends using the inorganic Hg BAF point estimate = 84 (rounded to 8×10^1) derived from the Niimi and Kissoon study.

I.2.2.7 Nickel

In aquarium tank studies, brown trout exposed to water containing radioactive nickel (^{63}Ni) showed the greatest accumulation of the metal in the gills, kidneys and liver, with relatively low accumulation in muscle tissue (Tjalve et al., 1988). The Ni concentration in muscle was related to the water concentration of Ni (Van Hoof and Nauwelaers, 1984). Similar to other cationic metals, increasing the acidity of water increases accumulation of Ni in fish.

A muscle BCF of 1.5 was recorded in the brown trout following 3 week exposure to Ni in a water tank. However, equilibrium of Ni between water and fish tissues had not been attained. Rainbow trout exposed to Ni in hard water (hardness = 320 mg $CaCO_3$/L) for six months accumulated little or no Ni in muscle tissue (BCF = 0.8-1.1) (Calamari et al., 1982).

In a field study, Nussey et al. (2000) calculated an average muscle tissue BAF of 19 in the moggel, a cyprinid fish, collected from a reservoir containing various heavy metals, including Ni, over a period of 15 months. Average muscle BAFs of 4 and 39 were calculated in common carp collected from two different wastewater treatment basins in Pennsylvania (Skinner, 1985). The acidity of the treatment basin water was not discussed, so it is unknown if water acidity played a role for the variation in BAF values.

In laboratory studies, accumulation of Ni in fish muscle tissues is relatively low compared to other inorganic metals discussed in this document. There are also relatively few published reports investigating fish bioaccumulation of Ni. Based on the BAFs from the two field studies by Nussey et al. (2000) and Skinner (1985), we calculated an arithmetic mean average BAF of 21 and recommend this value as a point estimate BAF for Ni.

I.2.2.8 Selenium

Selenium (Se) occurs in the environment in several oxidation states with different physicochemical and biological properties (Besser et al., 1993). Se from both natural and anthropogenic sources enters surface waters primarily as the highly soluble Se(IV) and Se(VI) oxidation states, which form selenite, SeO_3^{2-}, and selenate, SeO_4^{2-}, respectively. Organic selenides, Se(-II), including Se-amino acids and Se-proteins, methyl selenides, and other Se-substituted analogs of organosulfur compounds, are produced by biological reduction of selenite and usually occur at lower concentrations in water than inorganic Se species. Little information is available for organic selenides, so the BAF is based on total Se.

Se is an essential micronutrient for most aquatic organisms but is also toxic at relatively low environmental concentrations. It is reported that Se concentrations in fish muscle rarely exceed 1 ppm (wet weight) in the absence of exposure to Se from geologic sources or from industrial wastes (Cumbie and Van Horn, 1979).

Four-month exposure of juvenile bluegill and largemouth bass to selenite (Na_2SeO_3) in water resulted in BCF values of 288 and 153, respectively, and was independent of water temperature and hardness (Lemly, 1982). Accumulation of Se in muscle was relatively slow, reaching a steady-state concentration after 90 days of exposure in both fish species. Accumulation of Se in fish skeletal muscle was presumed to be a result of the high affinity of Se for sulfhydryl groups found on many organic molecules in muscle tissue. However, bioconcentration in muscle was quite low compared to BCF values for other organs and tissues. Lemly (1982) observed higher bioconcentration of Se in the spleen, heart, liver, kidney, gill, and erythrocytes.

In a food-chain study (algae-daphnids-bluegill), whole bluegill fry accumulated greater Se concentrations from food than from water in selenite-based exposures, and aqueous and food-chain Se bioaccumulation were approximately additive (Besser et al., 1993). However, in both aqueous and food-chain exposures based on selenite and selenate, Se bioaccumulation was greatest in algae and least in bluegills. Se concentrations in whole bluegill fry did not differ significantly between selenite and selenate treatments in either aqueous or food-chain exposures. Inorganic Se BCF values ranged from 13 to 106 in whole blue gill fry with 30- to 40-day exposures, although a steady-state concentration was not attained.

In a field study, Cumbie and van Horn (1979) analyzed muscle Se levels in various species of fish, primarily bluegill, other sunfish, carp and bullhead, during spring and summer from a reservoir with a high Se concentration. The range of muscle BAFs among all fish was 632 to 5450 with an arithmetic average of about 1780. Further research at the same reservoir observed muscle BAFs in warmwater sportfish (primarily various species of perch, catfish, sunfish and crappie) ranging from 739 to 2019 with an arithmetic average of 1351 (Lemly, 1985). There was evidence of biomagnification of Se through the food-chain, although when considering only muscle tissue of fish, levels of Se appeared to be similar to that of mulluscs, insects, annelids and crustaceans found at the reservoir.

Lower Se BAFs of 124 and 216 were calculated in muscle of white suckers and yellow perch, respectively, from an acidic lake in New York (Stripp et al., 1990). Based upon geochemistry, Se would be expected to be less soluble in acidic lakes. BAFs of 454 and 490 were determined for Se in muscle tissue of crappie and carp, respectively, collected from a wastewater treatment basin in Pennsylvania (Skinner, 1985).

The accumulation data indicate Se uptake from both food and water results in accumulation of Se in muscle tissue, and that BAF/BCF values can be quite variable even between different fish species within the same water body. The two related field studies investigating Se accumulation in fish from a North Carolina reservoir (Cumbie and Van Horn, 1979; Lemly, 1985) gave an average BAF of 1566 (1351 + 1780 / 2) combining all trophic level 3 and 4 fish. Not including the data from the acidic lake, we calculate an arithmetic mean BAF of 1019 when the average BAF from the North Carolina reservoir is combined with the average fish BAF from the Pennsylvania wastewater treatment basin from Skinner (1985). In support, the BAF is within the

predicted intervals (at water Se concentrations above 0.5 µg/L) of the Se whole fish bioaccumulation model for lentic systems developed by Brix et al. (2005). We recommend a default point estimate BAF of 1000 for selenium for use in the Hot Spots program.

I.3 Non-Bioaccumulated Chemicals

Some organic "Hot Spot" chemicals in which a significant airborne fraction can be found in the particle phase do not appear to be bioaccumulated in fish. For example, although data show that methylenedianiline (MDA) exists partly in the particle phase and is persistent in soils, the low log Kow of 1.59 (HSDB, 2008) and rapid metabolism in higher trophic level animals (ATSDR, 1998) indicate this chemical will likely not bioaccumulate in fish tissues. In addition, unpublished evidence summarized in ATSDR (1998) suggests that MDA does not bioaccumulate in carp. Until published evidence shows otherwise, a fish BAF for MDA will not be included in the fish pathway in the "Hot Spots" program.

In addition, OEHHA is proposing that fluoride should not be included in the fish pathway because fresh weight fluoride concentrations in muscle or the fillet portion of fish were found to be less than the water concentration, regardless of the weight of the fish (Gikunju, 1992; Mwaniki and Gikunju, 1995).

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Appendix J
Lactational Transfer

J.1 INTRODUCTION

Some toxic chemicals in the environment can accumulate in a woman's body and transfer to her milk during lactation. Chronic exposure to pollutants that accumulate in the mother's body can transfer a daily dose to the infant much greater than the mother's daily intake from the environment. For example, the mother's milk pathway can be responsible for about 25% of total lifetime exposure to dioxins and furans (USEPA, 2000).

Several reviews have listed numerous toxic chemical contaminants in human breast milk (Abadin et al., 1997; Liem et al., 2000; van Leeuwen and Malisch, 2002; LaKind et al., 2005; Li et al., 2009). Many of these chemical contaminants are carcinogens and/or have non-cancer health impacts on people who inhale or ingest them. Data suggest that ~~breast-fed~~ infants during the first two years of life have greater sensitivity to many toxic chemicals compared to older children and adults (OEHHA, 2009).

Multiple chemical contaminants have been measured in breast milk or have properties that increase their likelihood of partitioning to milk during lactation. OEHHA grouped these chemicals into the following four major categories:

1) Persistent highly-lipophilic, poorly metabolized organic contaminants, such as polychlorinated biphenyls (PCBs), polychlorinated dibenzofurans (PCDFs), and polychlorinated dibenzo-p-dioxins (PCDDs), are by far the most documented group. These, by virtue of their lipophilicity, are found almost entirely in the milk fat. PCBs, methyl sulfones, and hexachlorobenzene (HCB) methyl sulfones have also been measured in the lipid phase of breast milk.
2) Lipophilic but more effectively metabolized organic contaminants such as polycyclic aromatic hydrocarbons (PAHs) occur in breast milk. The PAHs are a family of over 100 different chemicals formed during incomplete combustion of biomass (e.g. coal, oil and gas, garbage, tobacco or charbroiled meat). Some of the more common parent compounds have been measured in breast milk and research suggests that chronic exposure to PAHs produces stores in maternal fat that can transfer (carryover) to breast milk (Fürst et al., 1993; Costera et al., 2009).
3) Inorganic compounds, metals, and some organo-metallics, including the heavy metals arsenic, lead, cadmium, and mercury, have been found in breast milk. These inorganics are generally found in the aqueous phase and most are bound to proteins, small polypeptides, and free amino acids. The lipid phase may also contain some organometallics (e.g. methyl mercury) and metalloids (such as arsenic and selenium).
4) Chemicals with relatively low octanol:water partition coefficients such as phenol, benzene, halobenzenes, halophenols, some aldehydes and the more polar metabolites of PCBs, PAHs, and pesticides may occur in both the aqueous and lipid phases of breast milk.

Since this document supports risk assessments conducted under the Air Toxics Hot Spots program, we are primarily discussing Hot Spots chemicals emitted from stationary sources.

Many of these persistent chemicals are ubiquitous in the environment and are global pollutants found in low concentrations in air, water and soil. Because some of these chemicals bio-concentrate in animal fat, the primary pathway of exposure to breastfeeding mothers would be consumption of animal products such as meat, milk, and eggs. Nearby polluting facilities can be a local source of exposure and can add to the mother's body burden of contaminants from global pollution through multiple pathways.

This appendix develops lactational transfer coefficients for use in estimating the concentration of a multipathway chemical in mother's milk from an estimate of chronic incremental daily dose to the mother from local stationary sources. OEHHA derived human lactation transfer coefficients from studies that measured contaminants in human milk and daily intake from inhalation or oral routes of exposure from global pathways (e.g. air, cigarette smoke or diet) in the same or a similar human population.

Briefly, human milk transfer coefficients (Tco_{hm}) represent the transfer relationship between the chemical concentration found in milk and the mother's chronic daily dose (i.e. concentration (µg/kg-milk)/dose (µg~~/kg~~/day) under steady state conditions. In its simplest form, the biotransfer factor is:

$$Tco_{hm} = C_m / D_t \qquad \text{(Eq. J-1)}$$

where:

Tco_{hm} = transfer coefficient from ingested and inhaled media (day/kg)
C_m = concentration of chemical in mother's milk (µg/kg-milk)
D_t = total maternal dose through all exposure routes (µg/day)

Equation J-2 estimates the concentration of contaminants in mother's milk by incorporating the Tco in the following way:

$$Cm = [DOSEair + DOSEwater + DOSEfood + DOSEsoil + DOSEdermal ~~D_inh + Dwi + Dfood + Dsi + Dderm~~] \times Tco_{hm} \times BW \qquad \text{(Eq. J-2)}$$

where:

BW = the body weight of the mother at age 25 (default = 70.7 kg)
DOSEair~~Dinh~~ = dose to the mother through inhalation (µg/kg-BW-day)
DOSEwater ~~Dwi~~ = dose to the mother though drinking water ingestion (µg/kg-BW-day)
DOSEfood~~Dfood~~ = dose to the mother through ingestion of food sources

(µg/kg-BW-day)
DOSEsoil~~Dsi~~ = dose to the mother through incidental ingestion of soil (µg/kg-BW-day)
DOSEdermal~~Dderm~~ = dose to the mother through dermal exposure to contaminated soil (µg/kg-BW-day)

However, if separate biotransfer information is available for the oral and inhalation route, equation J-3 incorporates route-specific Tcos in the following way:

$$Cm = [(D\_inh \times Tco_{m\_inh}) + (D\_ing \times Tco_{m\_ing})] \times BW \quad \text{Eq. J-3}$$

where:

D_ing = the sum of ~~dose~~ DOSEfood + DOSEsoil + DOSEwater through ingestion (~~µg~~mg/kg-BW-day)
D_inh = the sum of DOSEair + DOSEdermal through inhalation and dermal absorption (mg/kg-BW-day)
Tco_{m_inh} = biotransfer coefficient from inhalation to mother's milk (d/kg-milk)
Tco_{m_ing} = biotransfer coefficient from ingestion to mother's milk (d/kg-milk)

These coefficients, applied to the mother's chronic daily dose estimated by the Hot Spots exposure model, estimate a chemical concentration in her milk (see Table J.1-1).

Table J.1-1: Default Tcos (d/kg) for Mother's Milk

Chemical/chem. group	Tco	LCL	UCL
PCDDs - oral	3.7	2.68	5.23
PCDFs - oral	1.8	1.27	2.43
Dioxin-like PCBs - oral	1.7	0.69	4.40
PAHs – inhalation	1.55	0.731	3.281
PAHs – oral	0.401	0.132	1.218
Lead - inhalation	0.064	0.056	0.074

LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

Table J.1-1 lists the transfer coefficients for dioxins, furans, dioxin-like PCBs, PAHs and lead that OEHHA has estimated from data found in the peer-reviewed literature and reviewed in this appendix. One key factor that plays a role in the difference between oral and inhalation transfer coefficient (e.g., for -PAHs) is first pass metabolism which is lacking in dermal and inhalation exposures. Thus, for simplicity, OEHHA applies the transfer coefficients from inhalation to the dermal

absorption pathway for lead and PAHs. For lead, we are using the inhalation Tco for all the other pathways of exposure to the mother. Likewise, for PCDD/Fs and dioxin-like PCBs, we are using the oral Tco for the other pathways of exposure to the mother in Eq. J-2.

Estimates of toxicant biotransfer to breast milk are ideally chemical-specific. Data necessary to develop a transfer model are available in the open literature for a limited number of chemicals. Therefore, for some toxicants OEHHA has modeled the transfer of a class of chemicals with similar physical-chemical properties using a single Tco when data in the open literature are lacking.

The Hot Spots exposure model can estimate long-term total dose from an individual facility or group of facilities through many pathways of contamination and routes of exposure to the mother and ultimately to her infant. In this appendix, "multipathway toxicants" refers to airborne-released chemicals that can cause exposure through pathways in addition to inhalation. The indirect exposure pathways evaluated under the Hot Spots program include incidental ingestion of contaminated soil, ingestion of contaminated home-raised meat and milk, surface drinking water, homegrown produce, angler-caught fish and skin contact with contaminated soil.

Relative to the lifetime average daily dose to the infant from other exposure pathways in the Hot Spots exposure model, the dose of some chemicals from mother's milk will be negligible. However, the mother's milk pathway may be a substantial contributor to the estimated total lifetime cancer risk for some chemicals emitted from a Hot Spots facility. Exposure from global sources is expected to make up most (almost all) of a mother's toxicant body burden for chemcials like PCDDs. Therefore, the contribution to a mother's toxicant body burden from a single Hot Spot facility is expected to be very small. Regardless of the mother's toxicant body burden from both local and global sources, the benefits of breastfeeding outweigh the risks to the infant exposed to these toxicants during breastfeeding. Breast-feeding has a number of universally accepted benefits for the infant as well as for the mother (Mukerjee, 1998).

We established transfer coefficients (Tcos) for individual congeners of PCDDs/Fs and dioxin-like PCBs, individual and summary carcinogenic PAHs and lead through equations J.1-1 through J.1-3. We used data on exposure and breast milk contamination from background (global), accidental and occupational sources, and a set of simplifying assumptions. We assume that a mother's intake and elimination rates remain constant before lactation. We also assume that changes in a woman's body due to the onset of lactation occur as a single shift in elimination rate and do not change over the lactation period. Unless a study reported the geometric mean or median, we converted arithmetic mean and standard deviation to geometric mean and GSD.

In the following sections, we describe the methods for deriving specific Tcos from measurements of human milk intake and transfer estimates from studies of

populations published in the open literature. In some cases, OEHHA adjusted some measurements of human milk and contaminant intake to account for confounding factors. In such cases, OEHHA describes the method of adjustment in the text and table containing adjusted values.

J.2 Mothers' Milk Transfer Coefficients for PCDD/Fs and PCBs

Polychlorinated dibenzo-p-dioxins and furans (PCDD/Fs) are two series of almost planar tricyclic aromatic compounds with over 200 congeners, which form as impurities in the manufacture of other chemicals such as pentachlorophenol and PCBs. PCDD/Fs also form during combustion (e.g. waste incineration) and the breakdown of biomass (e.g. in sewage sludge and garden compost) (Liem et al., 2000). IARC has classified many dioxins and dioxin-like compounds as known or possible carcinogens (WHO, 1997; OEHHA, 2009). Their carcinogenic potency is related to the potency of 2,3,7,8-TCDD in a toxic equivalent (TEQ) weighting scheme (OEHHA, 2009).

The main exposure to PCDD/Fs in the general population from global sources is through the intake of food of animal origin. PCB exposure has been linked to fish consumption. For example, Jensen (1987) observed that congener distribution patterns in contaminated fish and human milk were very similar suggesting that one of the primary sources of human exposure to PCBs in the study population was ingestion of contaminated fish (Jensen, 1987).

Estimates of PCDD/F and PCB TEQ-intake from dietary sources contaminated by global sources can vary by 3 to 4-fold within some populations and by as much as 29-fold between populations (Liem et al., 2000; Focant et al., 2002). Exposure from diet can be at least an order of magnitude higher than intake from ambient air or cigarette smoking (i.e., 0.1 to 4 pg/day) (Liem et al., 2000).

J.2.1 Biotransfer of PCDD/Fs and PCBs to Human Milk

The potential health impacts from exposure to PCBs, PCDDs and PCDFs include carcinogenicity, developmental, endocrine disruption, reproductive toxicity, and neurotoxicity. These persistent, lipophilic compounds can accumulate in the fat of women, transfer to breast milk, and thus result in infant exposure. Some countries implemented measures to reduce dioxin emissions in the late 1980s (Liem et al., 2000). PCBs were banned in the late 1970's and are no longer used in commercial products. Nevertheless, following the PCB ban and efforts to reduce PCDDs, PCDFs emissions, these toxicants are still detected worldwide in human milk, although at declining levels.

The World Health Organization (WHO) has carried out a series of international studies on levels of approximately 29 dioxins and dioxin-like contaminants in breast milk. The first WHO-coordinated study took place in 1987-1988, the second round in 1992-1993 and the third round was initiated in 2000-2003. In the

second round, in which concentrations of PCBs, PCDDs and PCDFs were determined in milk samples collected in 47 areas from 19 different countries, mean levels in industrialized countries ranged from 10-35 pg I-TEQ/g-milk (Liem et al., 2000).

Much lower levels (40% lower than 1993) were detected in the 3rd round (Liem et al., 1995; Liem et al., 2000; van Leeuwen and Malisch, 2002) WHO exposure study. Nevertheless, several recent investigators have continued to measure levels of dioxin-like compounds in breast milk (LaKind et al., 2004; Barr et al., 2005; Wang and Needham, 2007; Li et al., 2009). PCBs still appear in human milk and are still much higher than the total concentrations of PCDDs and PCDFs. Several studies report pg/g-fat levels of PCDD/Fs compared to ng/g-fat levels of PCBs (100 to 1000 times higher) measured in human milk (Chao et al., 2003; Chao et al., 2004; Hedley et al., 2006; Sasamoto et al., 2006; Harden et al., 2007; Wittsiepe et al., 2007; Raab et al., 2008; Todaka et al., 2008).

Thus, nursing infants have the potential to ingest substantial doses during the breastfeeding period, relative to typical total lifetime dose of these compounds from global sources. Consequently, this pathway of exposure may supply a substantial fraction of PCDDs and PCDFs (about 25%) of the infant's total lifetime dose of these compounds (USEPA, 2000). Several studies have detected higher levels of PCBs in the sera (Schantz et al., 1994), adipose tissues (Niessen et al., 1984; Teufel et al., 1990) and bone marrow (Scheele et al., 1995) of mostly breast-fed children relative to partially breast fed infants. These studies were conducted many years after PCBs were banned and no longer used in commercial products. Some investigators have reported a 4-fold greater level of PCBs in the blood of fully breast-fed compared to partially breast-fed infants (Niessen et al., 1984).

In another study, Abraham et al (1994, 1996, 1998) measured elevated PCB concentrations in nursing infants after approximately one year of feeding (Abraham et al., 1994; Abraham et al., 1996; Abraham et al., 1998). These authors reported levels of 34 to 45 ppt (pg TEQ/g blood lipid) among breastfed infants versus 3 to 3.3 ppt blood lipid PCDD/F TEQ concentrations among formula fed infants.

Numerous studies have measured dioxins, furans and dioxin-like PCBs in mother's milk (Liem et al., 2000) The twenty nine dioxin-like PCBs listed in Table J.2-1 are recognized by OEHHA as carcinogens and have potency factors associated with them (OEHHA, 2008). Concentrations of TCDD (2,3,7,8-tetrachlorodibenzo-p-dioxin), the most toxic PCDD, are low relative to other PCDDs and more than 50% of the total PCDD content consists of Octa-CDD. Early studies found around 70% of the total Hexa-CDDs (HxCDDs) is 1,2,3,6,7,8-HxCDD, and the remainder is mainly 1,2,3,4,7,8-HxCDD and 1,2,3,7,8,9-HxCDD (USEPA, 1998). These proportions have not shifted in recent studies (Sasamoto et al., 2006; Zhao et al., 2007; Raab et al., 2008).

PeCDD (1,2,3,7,8 Penta-CDD) is always found in the emissions from waste incinerators (USEPA, 1998). Early studies indicated that the presence of 1,2,3,7,8-PeCDD with other PCDDs/PCDFs in human milk suggested that the major source of exposure came from waste incinerator emissions (Buser and Rappe, 1984; Rappe et al., 1985; Mukerjee and Cleverly, 1987). Note that these congeners are measurable in human milk currently (Sasamoto et al., 2006; Zhao et al., 2007; Raab et al., 2008).

Levels of PCDFs in human milk tend to be lower than PCDDs. However, PCDFs dominate in particulates emitted by combustion sources, including hazardous waste incinerators, and are present in higher concentrations in the atmosphere than PCDDs (USEPA, 1998). HxCDDs/HxCDFs and HpCDDs/HpCDFs are prevalent in pentachlorophenol. Incineration of wood and other products impregnated with pentachlorophenol results in the formation of these congeners and emissions of hexa- and hepta-CDDs/CDFs. Both 1,2,3,7,8 and 2,3,4,7,8-PeCDFs have been detected in human milk, but 90% of the PeCDFs is generally 2,3,4,7,8-PeCDF. 1,2,3,4,7,8- , and 1,2,3,6,7,8- HxCDFs. 2,3,4,6,7,8-HxCDFs, and1,2,3,4,6,7,8-HpCDF are also prevalent.

Several investigators have observed that dose, degree of chlorination, degree of lipophilicity, and molecular weight influence how much PCDD/F congener is absorbed through the lungs or gut, metabolized and transferred from blood to milk (Yakushiji, 1988; Abraham et al., 1998; Schecter et al., 1998; Kostyniak et al., 1999; Oberg et al., 2002; Wittsiepe et al., 2007).

Numerous studies have attempted to correlate exposure to individual dioxins, furans and dioxin-like PCBs from ingestion of contaminated food with levels in human biological samples such as blood and milk. Transfer from intake sources to human milk has often been estimated in the context of accidental or occupational exposures or after a substantial decline in environmental concentrations (Liem et al., 1995; Pinsky and Lorber, 1998; Liem et al., 2000; Focant et al., 2002; Furst, 2006; Milbrath et al., 2009). Steady state conditions are not reached in these studies because the half-lives of these compounds are in years and exposure changed considerably over the period evaluated in each study.

Others have attempted to model the relationship between maternal intake and concentration in mother's milk using an indicator compound such as TCDD (Smith, 1987; Lorber and Phillips, 2002). Less understood is the relationship between modeled and measured transfer estimates of individual dioxins, furans and dioxin-like PCBs. The following sections describe the sources of data and methods for deriving estimates of transfer for an array of dioxins, furans and dioxin-like PCBs that have accounted to some extent for the non-steady state condition and other confounders.

J.2.2 Oral Biotransfer

OEHHA located a series of studies conducted on the Dutch population that allows for an oral biotransfer estimate of dioxins and furans, and accounts for changing exposure conditions. In 1988, Albers et al. collected and analyzed three hundred nineteen breast milk samples from women enrolled through 28 maternity centers located throughout the Netherlands. Maternity centers were selected based on geographic distribution and degree of urbanization. Human milk samples were analyzed for 17 PCDD/F congeners and 8 PCB congeners (Albers et al., 1996).

Liem et al. (1995) took a similar approach to collect about 100 samples from first-time mothers enrolled in 1993 through maternity centers dispersed throughout The Netherlands. Based on information obtained from a questionnaire about characteristics of the study subject, investigators determined that the 1993 cohort appeared to be comparable to the cohort studied in 1988. With one exception, (1,2,3,4,7,8- HxCDD), a consistent downward trend can be seen among congeners of PCDD/Fs and PCB-118 that were analyzed during both sampling periods, (Table J.2-1).

Table J.2-1: Summary Estimates of Dioxin-like Compounds Dietary Intake during Three Periods Over 15 years, and Human Milk Levels over Five Years in the Dutch Population

Chemical/ group	**TEF**	**1978 (diet)[a]**	**1984/5 (diet)[a]**	**1994 (diet)[a]**	**1988 (milk)[b]**	**1993 (milk)[a]**
		Mean, SD	**Mean, SD**	**Mean, SD**	**Mean, SD**	**Mean, SD**
		pg/d*	**pg/d***	**pg/d***	**pg/kg-milk**	**pg/kg-milk**
2,3,7,8-TCDD	1	13.2, 1.32	6, 2.94	3.6, 1.26	264,14	124, 56
1,2,3,7,8-PeCDD	1	39.6, 6.73	15, 4.65	4.8, 2.26	435,185	324, 116
1,2,3,4,7,8-HxCDD	0.1	85.8, 23.17	23.4, 17.55	7.2, 5.98	328,51	344, 192
1,2,3,6,7,8-HxCDD	0.1	325.8, 45.61	89.4, 42.02	19.8, 22.77	2445,349	1484, 668
1,2,3,7,8,9-HxCDD	0.1	105, 21.0	32.4, 21.38	10.8, 9.61	395,32	276, 132
1,2,3,4,6,7, 8-HpCDD	0.01	2016, 463.68	1908, 2671.2	150, 120	3242,114	1796, 984
OctaCDD	0.0001	12420, 4595	9180, 10281	1170, 749	28844,2896	11788, 6708
2,3,7,8-TCDF	0.1	106.8, 9.61	84, 31.08	21, 14.7	100,8	16, 16
1,2,3,7,8-	0.05	24.6, 4.67	6.6, 2.71	3.6, 1.51	30,10	8, 8

Chemical/ group	TEF	1978 (diet)[a]	1984/5 (diet)[a]	1994 (diet)[a]	1988 (milk)[b]	1993 (milk)[a]
		Mean, SD	Mean, SD	Mean, SD	Mean, SD	Mean, SD
		pg/d*	pg/d*	pg/d*	pg/kg-milk	pg/kg-milk
PeCDF						
2,3,4,7,8-PeCDF	0.5	178.8, 25.03	65.4, 13.73	23.4, 12.87	807,108	720, 300
1,2,3,4,7,8-HxCDF	0.1	178.8, 30.40	43.8, 9.20	27.6, 11.04	293,20	208, 92
1,2,3,6,7,8-HxCDF	0.1	54, 3.78	27, 6.21	13.8, 5.52	261,17	176, 84
1,2,3,7,8,9-HxCDF	0.1	<0.05	<0.04	<0.04	NA	NA
2,3,4,6,7,8-HxCDF	0.1	55.8, 6.70	25.2, 6.80	9, 5.76	133,19	96, 52
1,2,3,4,6,7,8-HpCDF	0.01	471, 117.75	176.4, 65.27	51.6, 22.19	523,55	240, 124
1,2,3,4,7,8,9-HpCDF	0.01	39, 4.68	7.8, 5.07	3, 1.62	NA	4, 4
OctaCDF	0.0001	466.8, 107.36	195, 78.0	69.6, 37.58	49,10	12, 12
PCB-77	0.0001	NA	NA	NA	NA	452, 872
PCB-81	0.0001	NA	NA	NA	NA	NA
PCB-126	0.1	1350, 202.5	924, 221.76	378.6, 87.08	NA	3284, 1448
PCB-169	0.01	270, 54.0	181.2, 86.98	174, 214.02	NA	2320, 988
		ng/d*	ng/d*	ng/d*	ng/kg-milk	ng/kg-milk
PCB-105	0.0001	71.4, 13.57	70.2, 33.7	13.2, 5.54	NA	160, 80
PCB-114	0.0005	6.6, 0.92	11.4, 8.66	1.8, 1.35	NA	NA
PCB-118	0.0001	289.2, 43.38	247.2, 111.24	49.2, 15.25	1009,565	971.2, 456
PCB-123	0.0001	18.6, 3.91	15, 7.65	2.4, 0.89	NA	NA
PCB-156	0.0005	191.4, 63.16	27.6, 8.28	9, 2.79	NA	564, 236
PCB-157	0.0005	22.2, 6.44	4.8, 1.73	1.8, 0.72	NA	108, 48
PCB-167	0.00001	79.2, 22.18	11.4, 2.51	3.6, 1.01	NA	152, 64
PCB-189	0.0001	43.8, 13.14	2.4, 0.53	1.2, 0.31	NA	48.4, 48

[a] (Liem et al., 2000); [b] (Albers et al., 1996), NA, not available; * Conversion from g-fat to kg-milk = 0.04 g-fat/g-milk*1000g/kg; Liem et al. reported dietary intake

estimates in units of mass/body weight/day. Therefore, we converted their estimates to units of mass/day by multiplying by the default 60 kg body weight used by Liem et al (Liem et al., 2000).

Liem et al. (2000) reported dietary intake for three time-periods (see Table J.2-1)(Liem et al., 2000). Dietary intake estimates were based on concentrations of PCDD/Fs and PCBs measured in composite samples of 24-hr duplicate diets in the Dutch adult population in 1978, 1984-85, and 1994 and combined with individual consumption data collected in 1987-1988 (Albers et al., 1996) (briefly summarized previously) for approximately 6000 individuals from 2200 families over a 2-day period . In a separate study, these same investigators estimated dioxin and dioxin-like compounds in human milk fat collected in the period 1992-1993 from more than 80 women (Liem et al., 1995; Liem et al., 2000).

Liem et al. (2000) observed a downward trend in estimated dietary intake of individual congeners of PCDDs PCDFs and PCBs in the Dutch population during three intervals from 1978 to 1994 (see Table J.2-1)(Liem et al., 2000). A downward trend was also seen in a study of these toxicant levels in the diet and human milk of the German population from 1983 - 2003 (Furst, 2006; Wilhelm et al., 2007). However, about half of the mono-ortho PCBs did not show a similar linear decline. This pattern is consistent with observations made by Alcock et al., (1996) who reported some evidence that the environmental load of PCDD/Fs increased in the 1960s, peaked around 1975 and then began to decline (Alcock et al., 1996).

OEHHA has derived lactational transfer coefficients for PCDD/Fs and dioxin-like PCBs from studies of exposure from global sources and by multiple pathways. The proportional contribution from various exposure pathways to total exposure from a single Hot Spots facility is likely to be quite different from that found from global sources. However, we assume that the estimate of transfer to milk from global sources, such as that derived from the Dutch studies, reasonably represents the transfer in persons from communities near Hot Spot facilities in California.

The Hot Spots program allows for reporting emissions of individual congeners of dioxins, furans and PCBs, when emissions are speciated. It also permits reporting of emissions as total dioxins and furans or PCBs. Speciation of emissions produces a more accurate (and lower) risk estimate. This is because unspeciated emissions are assumed to be 2,3,7,8-TCDD, which has the highest potency factor among the dioxins and furans. Therefore, OEHHA has derived congener Tcos for individual PCBs and dioxins that can be used when emissions are speciated.

J.2.3 Mothers' Milk Transfer Coefficients (Tco) for PCDD/Fs and PCBs

To calculate oral Tcos, OEHHA used adjusted reference half-lives for the chemicals in adults estimated from dietary and occupational exposures. OEHHA estimated oral Tcos for these chemicals using estimates of body weight reported in Chapter 10 of this document, reference half-lives reported in Milbrath et al. (2009) and the steady-state equation developed by Smith (1987) (Smith, 1987; Milbrath et al., 2009).

Milbrath et al., (2009), in a systematic review of studies reporting half-lives in the human body, developed average human biological reference half-lives for 28 out of 29 dioxins and dioxin-like PCBs with OEHHA-recognized potency factors (see Table J-2-2) (Milbrath et al., 2009).

Table J.2-2: Half lives of PCDD/Fs and Dioxin-like PCB Congeners in Humans as Measured in Blood (Milbrath et al., 2009)

Chemical	N studies	Half life range (yrs)	Mean half life in adult (yrs)	Median half life in adult (yrs)	Study
TCDD	10	1.5 – 15.4	7.2	6.3	a
1,2,3,7,8-PeCDD	4	3.6 – 23.1	11.2	8.5	a
1,2,3,4,7,8-HxCDD	3	1.4 – 19.8	9.8	10.9	a
1,2,3,6,7,8-HxCDD	4	2.9 – 70	13.1	12	a
1,2,3,7,8,9-HxCDD	3	2.0 – 9.2	5.1	6.8	a
1,2,3,4,6,7,8-HpCDD	4	1.6 – 16.1	4.9	3.7	a
OctaCDD	4	1.8 - 26	6.7	5.7	a
2,3,7,8-TCDF	1	0.4	2.1	0.9	b
1,2,3,7,8-PeCDF	4	0.9-7.5	3.5	1.9	b
2,3,4,7,8- PeCDF	16	1.5-36	7	4.9	b
1,2,3,4,7,8-HxCDF	14	1.5-54	6.4	4.8	a
1,2,3,6,7,8-HxCDF	6	2.1-26	7.2	6	a
2,3,4,6,7,8-HxCDF	6	1.5-19.8	2.8	3.4	b
1,2,3,4,6,7,8-HpCDF	11	2.0-7.2	3.1	3	a
1,2,3,4,7,8,9-HpCDF	1	2.1-3.2	4.6	5.2	b
OctaCDF	1	0.2	1.4	1.6	b
PCB-77	2	0.1-5.02	0.1	0.1	c
PCB-81	-	-	0.7	0.73	c
PCB-126	3	1.2-11	1.6	2.7	c
PCB-169	3	5.2-10.4	7.3	10.4	c
PCB-105	4	0.56-7.0	2.4	2.4	c
PCB-114	2	7.4-31.7	10	25	c
PCB-118	10	0.82-33.7	3.8	1.6	c
PCB-123	2	5.3-15.3	7.4	12	c
PCB-156	7	1.62-100	16	5.35	c
PCB-157	2	13-26	18	20	c
PCB-167	2	8.7-35	12	12	c
PCB-189	2	16-166.7	22	41	c

[a] (Flesch-Janys et al., 1996); [b] (van der Molen et al., 1996); [c] (Ogura, 2004)

Each reference half-life was derived from data on occupational exposures (Flesch-Janys et al., 1996; van der Molen et al., 1996) or dietary intake of the general population (Ogura, 2004). Note that mean half-lives vary by more than 2-fold among dioxin, 5-fold among furans and more than 100-fold among PCB congeners.

In an initial review of the literature, Milbrath et al (2009) reviewed evidence about factors that can affect elimination rates. Personal factors such as body fat, smoking status and past lactation practices can affect body burden and elimination rates. For example, smoking has been associated with a 30% to 100% increase in elimination rates of some dioxin congeners (Flesch-Janys et al., 1996; Milbrath et al., 2009). As well, the onset of lactation sets a new elimination pathway into effect and can substantially reduce the maternal body burden of PCBs during 6 months of lactation (Niessen et al., 1984; Landrigan et al., 2002).

Half-lives derived from children would be less than that from older adults due, in part, to the effects of the growing body on estimates of blood concentrations. Models based on rat data demonstrate a linear relationship between increasing fat mass and half-life length at low body burdens, with the impact of adipose tissue on half-life becoming less important at high body burdens (Emond et al 2006). At high body burdens, dioxins are known to up-regulate the enzymes responsible for their own elimination. Human data suggest that the serum concentration of TCDD where this transition occurs is 700 pg/g and 1,000 – 3,000 pg/g for PCDFs (Kerger et al 2006, Leung et al 2005). Therefore, investigators selected a subset of data based on the following criteria:

- blood serum concentrations of PCDD/Fs were less than 700 pg /g blood lipid total toxic equivalents (TEQs) at the time of sampling
- subjects were adults
- measurements were not reported as inaccurate in later studies

Milbrath et al selected the reference values to represent a 40- to 50-year-old adult with blood dioxin concentrations in the range where fat drives the rate of elimination (i.e. at lower body burdens). In addition, Milbrath rejected half-lives longer than 25 years if the original study calculated half-lives assuming steady-state conditions.

For the retained subset, the investigators calculated the mean and range of half-lives to establish a representative set of half-lives for each congener in a moderately exposed adult (Milbrath et al., 2009). They also adjusted reference half-lives for age, body fat, smoking habits and breast-feeding status as these factors were all strong determinants of half-life in humans (Milbrath et al., 2009).

A generally accepted approach to estimating the concentration of a lipophilic chemical in milk is outlined by Smith (1987). This approach is based on average

maternal daily intake, an estimate of the half-life ($t_{1/2}$) of PCDDs/PCDFs and PCBs and body weight-normalized (BW) proportionality factors. The chemical concentration in breast milk can be calculated by equation J-4:

$Cm = (Emi)(t_{1/2})(f1)(f3)/(f2)(0.693)$ Eq. J-4

Cm = chemical concentration in milk (mg/kg milk)
Emi = average daily maternal intake of contaminant (mg/kg-BW/day)
$t_{½}$ = biological half-life (days)
f1 = proportion of chemical in mother that partitions into fat (e.g. 0.8)
f2 = proportion of mother's body weight that is fat (e.g. 0.33 = kg-fat/kg-BW)
f3 = proportion of breast milk that is fat (e.g., 0.04 = kg-fat/kg-milk)

Smith's approach requires an estimate of the biological half-life of PCBs and PCDDs/PCDFs in the adult human and is restricted to poorly metabolized, lipophilic chemicals that act predominantly by partitioning into the fat component and quickly reaching equilibrium in each body tissue (including breast milk).

Because of Milbrath's approach, Tco-estimates for dioxins, furans and dioxin-like PCBs apply the following conservative assumptions regarding factors that affect elimination rates:

- lower enzyme induction based on nonsmokers with a body burden below 700 ppt in the blood
- adult age
- no recent history of breast-feeding
- body fat estimates based on older adults

Transfer coefficients (Ng, 1982) are ideally calculated from the concentration of contaminant in milk following relatively constant long-term exposure that approximates steady state conditions. Because Smith's equation is linear, it can be rearranged to solve ratio of the chemical concentration in milk to the chemical taken into the body per day, which is the transfer coefficient (Equation J-5).

$Tco = Cm/(Cf)(I)$ Eq J-5

Tco is the transfer coefficient (day/kg or day/liter)
Cm = measured chemical concentration in milk (µg/kg or mg/liter milk)
Cf = measured chemical concentration in exposure media (e.g. food) (µg/kg food)
I = reported daily intake of exposure media (kg/day of food)

The following equation (Eq-J-6) is equation Eq J-5 substituted into equation Eq J-4 and rearranged to solve for Tco.

$Tco = (t_{1/2})(f1)(f3)/(BW)(f2)(0.693)$ Eq J-6

Note that Emi in equation J-4 = (Cf)(I)/BW with units of mg/kg-BW/day. BW is the average adult body weight of the mother (kg).

Transfer coefficients (Tcos) in Table J.2-3 (column-2) combine milk data (milk concentration of PCDD/Fs and PCBs) with dietary intake estimates listed in Table J.2-1. OEHHA derived individual Tcos from data presented in (Liem et al., 1995; Albers et al., 1996; Liem et al., 2000). Because the median is a reasonable estimate of the geometric mean in skewed distributions, Tcos were derived from median half-lives listed in column-5 of Table J.2-2. Tcos range from less than one to more than ten d/kg-milk among dioxins and furan and less than two to more than 20 d/kg-milk among dioxin-like compounds.

Table J.2-3: Arithmetic Mean Transfer Coefficients (Tcos) for Individual PCDD/F and PCB Congeners Measured in Human Milk and Dietary Intake from a Dutch Population (d/kg-milk) Compared to the Median and Geometric Mean Tcos Derived from Reference Half-lives ($t_{1/2}$) and Equation J-6

Chemical/group	Tcos (GM) based on slope factors	Tco based on median reference half life (Milbrath et al 2007)	Tco based on t1/2 GM*	Tco based on t1/2 GSD	Tco based on t1/2 LCL	Tco based on t1/2 UCL
2,3,7,8-TCDD	49.62	5.36	4.02	2.76	2.14	7.53
1,2,3,7,8-PeCDD	8.76	7.24	6.53	2.16	3.07	13.90
1,2,3,4,7,8-HxCDD	0.98	9.28	5.60	3.41	1.40	22.48
1,2,3,6,7,8-HxCDD	11.02	10.21	3.27	4.20	0.80	13.32
1,2,3,7,8,9-HxCDD	4.89	5.79	3.32	1.91	1.60	6.88
1,2,3,4,6,7,8-HpCDD	2.88	3.15	1.96	2.74	0.73	5.26
OctaCDD	5.54	4.85	2.29	3.25	0.72	7.28
2,3,7,8-TCDF	3.18	0.77	1.76	1.36	0.96	3.23
1,2,3,7,8-PeCDF	3.43	1.62	1.91	2.49	0.78	4.68
2,3,4,7,8- PeCDF	2.77	4.17	1.78	4.24	0.88	3.62
1,2,3,4,7,8-HxCDF	2.16	4.09	0.99	5.29	0.41	2.38
1,2,3,6,7,8-HxCDF	7.89	5.11	2.64	3.01	1.09	6.39
1,2,3,7,8,9-HxCDF	NA	NA	NA	NA	NA	NA
2,3,4,6,7,8-HxCDF	3.18	2.89	0.55	3.18	0.22	1.39
1,2,3,4,6,7,8-HpCDF	2.40	2.55	1.82	1.63	1.36	2.44
1,2,3,4,7,8,9-HpCDF	NA	4.43	3.63	1.34	2.06	6.42
OctaCDF	0.32	1.36	0.99	2.83	0.13	7.55
PCB-77	NA	NA	0.06	6.38	0.004	0.72
PCB-81	NA	NA	0.38	1.35	0.248	0.57

Chemical/group	Tcos (GM) based on slope factors	Tco based on median reference half life (Milbrath et al 2007)	Tco based on t1/2 GM*	Tco based on t1/2 GSD	Tco based on t1/2 LCL	Tco based on t1/2 UCL
PCB-126	NA	2.30	0.34	2.61	0.11	1.01
PCB-169	NA	8.85	5.60	1.27	4.28	7.32
PCB-105	NA	2.04	1.07	3.02	0.36	3.16
PCB-114	NA	2.04	2.74	3.11	0.57	13.20
PCB-118	0.01	1.36	0.55	6.17	0.18	1.70
PCB-123	NA	1.36	2.93	2.63	0.77	11.18
PCB-156	NA	4.55	3.23	7.10	0.76	13.81
PCB-157	NA	17.02	14.10	1.21	10.84	18.34
PCB-167	NA	10.21	5.93	1.76	2.70	13.00
PCB-189	NA	34.90	4.23	2.77	1.03	17.33

slope factors obtained from the longest interval between measures of diet (1978-1994) and milk (1988-1993) in the Dutch population; * GM, geometric mean, GSD, geometric standard deviation derived from natural log of three half-life values, low, high and median reported in Milbrath et al. (Milbrath et al., 2009) LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

OEHHA evaluated the relationship between Tcos predicted by Equation J-6 (column 3) using median reference half-lives and those derived from slope factors (column 2). Briefly, slope factors were calculated by taking the difference between cross-sectional dietary intake estimates taken in 1978 and 1994 and the difference between cross-sectional human milk concentrations taken in 1988 and 1993 from the Dutch population. Most Tcos derived from reference half-lives compare reasonably well with those derived from slope factors.

In columns 4-7 of Table J.2-3 the GM, GSD and 95%CLs of transfer coefficients (Tcos) for individual dioxins and dioxin-like congeners are derived from equation J-6 and geometric distribution estimates and 95% confidence intervals of half-lives provided in (Milbrath et al., 2009).

A Random-effects model derived summary estimates shown in Table J.2-4 from individual summary estimates shown in columns 4-7 of Table J.2-3.

Table J.2-4: Tco Estimates Stratified by Dioxin, Furan and Dioxin-like PCB Congeners (mean, 95%CI from Random-effects Model)

Chemical group	N congeners	Tco	LCL	UCL
PCDDs - oral	7	3.7	2.68	5.23
PCDFs - oral	9	1.8	1.27	2.43
Dioxin-like PCBs - oral	12	1.7	0.69	4.40

LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

OEHHA believes that a Random-effects model is appropriate because OEHHA assumes that the compounds found in exposure studies are a subgroup from a population of congeners in each subgroup (i.e., dioxins and dioxin-like compounds). Random-effects models assume there are multiple central estimates and incorporate a between-compound estimate of error as well as a within-compound estimate of error in the model. In contrast, a Fixed-effects model assumes that observations scatter about one central estimate (Kleinbaum, 1988).

J.2.4 Carryover Rate

Looking at mother's milk Tcos in terms of carryover rate suggests that accumulation of dioxins and dioxin-like compounds in the mother's body occurs but varies by more than 100-fold among individual compounds (based on Tcos derived from equation J-6).

Carryover rate, a term commonly used in the dairy literature (McLachlan et al., 1990) is defined as the daily output of dioxins and dioxin-like compounds in mother's milk (µg/day) over the daily intake of dioxins and dioxin-like compounds (µg/day). This rate is estimated by multiplying a dioxin's and dioxin-like Tco by the daily output of mother's milk. Since milk production in human mothers are about 1.0 kg/day, a dioxins and dioxin-like Tco is the carryover rate for a typical 60 kg woman.

A carryover rate > 1 would suggest that dioxins and dioxin-like compounds could accumulate in body fat and transfer to the fat in mother's milk. With an average dioxin Tco of 3.7 d/kg, 370% of the mother's average daily intake from ingested sources, transfers to mother's milk. This high transfer-value suggests that accumulation or concentrating of carcinogenic dioxins and dioxin-like compounds occur in the mother's body. Oral Tcos less than one d/kg (e.g., 1,2,3,4,7,8-HxCDF and 2,3,4,6,7,8-HxCDF) suggest that net metabolism or excretion occurs in the mother's body.

J.3 Mothers' Milk Transfer Coefficients for PAHs

The polycyclic aromatic hydrocarbons (PAHs), a family of hundreds of different chemicals, are characterized by fused multiple ring structures. These compounds are formed during incomplete combustion of organic substances (e.g. coal, oil and gas, garbage, tobacco or charbroiled meat). Thus, PAHs are ubiquitous in the environment and humans are likely to be exposed to these compounds on a daily basis. PAHs are a common pollutant emitted from Hot Spots facilities and are evaluated under the program.

Only a small number of the PAHs have undergone toxicological testing for cancer and/or noncancer health effects. PAHs with cancer potency factors are the only ones that can be evaluated for cancer risk using risk assessment. However, PAHs that lack cancer potency factors have been measured in various studies

and can serve as a useful surrogate for PAHs with cancer potency factors because of their physical-chemical similarity to PAHs with cancer potency factors.

Less than 30 specific PAHs are measured consistently in biological samples or in exposure studies. For example, Table J.3-1 lists commonly detectable PAHs in food and the environment (Phillips, 1999). In one analysis, pyrene and fluoranthene together accounted for half of the measured PAH levels in the diet (Phillips, 1999). Table J.3-1 includes nine PAHs that have cancer potency factors and are recognized by OEHHA as presenting a carcinogenic risk to humans (OEHHA, 2009).

Table J.3-1: PAHs with and without Cancer Potency Factors Commonly Measured in Food (Phillips, 1999)

PAHs without Cancer Potency Factors	PAHs with Cancer Potency Factors
Benzo[ghi]perylene	Dibenz[a,h]anthracene
Fluoranthene	Indeno[1,2,3-cd]pyrene
Pyrene	Benzo[a]pyrene
Phenanthrene	Benzo[k]fluoranthene
Anthracene	Chrysene
Fluorene	Benzo[b]fluoranthene
Acenaphthylene	Benz[a]anthracene
Acenaphthene	Naphthalene
Benzo[b]naphtho[2,1-d]thiophene	Benzo[j]fluoranthene
Benzo[ghi]fluoranthene	
Cyclopenta[cd]pyrene	
Triphenylene	
Perylene	
Benzo[e]pyrene	
Dibenz[a,j]anthracene	
Anthanthrene	
Coronene	

Few investigators have attempted to correlate PAH exposure from contaminated food and ambient air with PAH concentrations in human biological samples such as the blood or mother's milk. This is likely due to insensitive limits of detection for PAHs yielding few positive measurements, possibly due to the rapid and extensive metabolism of PAHs in mammals (West and Horton, 1976; Hecht et al., 1979; Bowes and Renwick, 1986).

This extensive metabolism often results in low or immeasurable concentrations of PAHs in mother's milk and blood (e.g. (Kim et al., 2008)). Nevertheless, emissions of PAHs from stationary sources are common and the increased

sensitivity of infants to carcinogens necessitates looking into development of mother's milk transfer factors (Tco) for carcinogenic PAHs.

Four studies have measured PAHs in mother's milk of smokers and non-smokers (see Table J.3-2). The 16 PAHs reported in these studies are among the most common PAHs released into the environment and found in biological samples (Phillips, 1999; Ramesh et al., 2004).

TABLE J.3-2: Measured Concentrations (µg/kg-milk) of PAHs in Human Milk

Chemical / chemical group	Urban smokers (Italy) n=11[a] (Zanieri et al., 2007)	Urban non-smokers (Italy) n=10 (Zanieri et al., 2007)	Rural Non-smokers (Italy) n=11 (Zanieri et al., 2007)	Rural Non-smokers (Italy) n=10 (Del Bubba et al., 2005)	Non-smokers (USA) n=12 (Kim et al., 2008)	Un-known (Japan) n=51 (Kishikawa et al., 2003)
PAHs with Cancer Potency Factors AM, SD						
Naphthalene	10.54, 6.08	6.83, 2.18	4.42, 1.17	4.70, 2.44	NA[d]	NA
Chrysene	0.90, 2.09	0.59, 0.94	<0.018	<0.018	--[c]	0.06, 0.08
Benzo[a] anthracene	0.98, 1.47	0.61, 0.94	0.07, 0.16	0.974, 1.82	--	0.004, 0.01
Benzo[b] fluoranthene	0.53, 1.24	0.55, 0.80	<0.019	0.560, 1.39	--	0.41, 0.26
Benzo[k] fluoranthene	0.13, 0.30	<0.018	<0.018	0.114, 0.343	--	0.01, 0.01
Benzo[a]pyrene	0.52, 0.65	<0.018	<0.018	<0.018	--	0.002, 0.003
Dibenzo[a,h] anthracene	1.33, 3.33	<0.014	<0.014	<0.014	--	0.01, 0.01
Indeno[1,2,3-c,d] pyrene	0.42, 0.94	<0.011	<0.011	<0.011	--	0.003, 0.01
Sum	15.35	8.58	4.5	6.4	--	0.5
PAHs without Cancer Potency Factors AM, SD						
Anthracene	0.16, 0.45	0.71, 1.57	0.21, 0.56	0.616, 1.58	--[c]	0.01, 0.01
Acenaphthylene	7.73, 11.95	9.09, 3.08	4.11, 3.62	6.95, 4.18	NA[d]	NA
Phenanthrene	3.67, 2.39	0.97, 0.51	0.64, 0.58	0.553, 0.493	0.49, 0.44	0.25, 0.16
Fluorene	5.13, 9.45	1.50, 1.60	0.06, 0.21	1.06, 1.70	0.13, 0.13	NA

TABLE J.3-2: Measured Concentrations (µg/kg-milk) of PAHs in Human Milk

Chemical / chemical group	Urban smokers (Italy) n=11[a] (Zanieri et al., 2007)	Urban non-smokers (Italy) n=10 (Zanieri et al., 2007)	Rural Non-smokers (Italy) n=11 (Zanieri et al., 2007)	Rural Non-smokers (Italy) n=10 (Del Bubba et al., 2005)	Non-smokers (USA) n=12 (Kim et al., 2008)	Un-known (Japan) n=51 (Kishikawa et al., 2003)
Acenaphthene	10.55, 17.73	3.12, 1.79	1.37, 1.31	2.72, 1.69	NA	NA
Pyrene	1.03, 1.25	1.40, 3.01	0.21, 0.30	0.620, 1.64	0.05, 0.04	0.02, 0.05
Fluoranthene	2.86, 2.60	0.54, 0.76	0.53, 1.03	0.250, 0.441	0.06, 0.05	0.02, 0.03
Benzo[g,h,i] perylene	1.51, 2.24	<0.018	<0.018	<0.018	--	--
Sum	32.64	17.33	7.13	12.8	0.73	0.3

[a] group includes one rural smoker; [b]values below detection limits were treated as zero in estimates of the mean; [c] – indicates all measurements were below the detection limits; [d] not assessed; (Kishikawa et al., 2003; Del Bubba et al., 2005; Zanieri et al., 2007; Kim et al., 2008) µg, microgram; kg, kilogram; n, number of samples; AM, Arithmetic Mean; SD, Standard Deviation

In this section, we estimated Tcos for PAHs with and without cancer potency factors. Additionally, none of the PAHs has a chronic Reference Exposure Level (REL) value. PAHs without cancer potency factors (other) are included because they:

- have structures similar to carcinogenic PAHs and are thus suitable as surrogate compounds
- are frequently measured in exposure studies
- produce measurements at detectable levels

In Table J.3-2, the sum of carcinogenic PAHs in human milk of Italian women is about 2-fold lower than the sum of other PAHs.

Because of their similarities in structure, the Tcos developed from other abundant PAHs are expected to compare reasonably well with the Tcos developed for less abundant carcinogenic PAHs.

J.3.1 Inhalation Biotransfer of PAHs to Mother's Milk

Biotransfer of PAHs to breast milk via the mother's inhalation pathway must be considered separately from biotransfer of PAHs to breast milk from the mother's oral route. PAHs will show a different pattern of metabolism depending on the route of exposure because of first pass metabolism in the liver from oral exposure, different rates and patterns of metabolism in the lung, and other factors. Smoking cigarettes represents a significant source of PAHs resulting in measurable levels of PAHs in mother's milk. Therefore, OEHHA chose a study that measured PAH concentrations in breast milk in smoking women and nonsmoking women to estimate inhalation Tcos for PAHs.

Of the four studies listed in Table J.3-2, the Italian study by Zanieri et al. (2007) allowed correlation of PAH intake via chronic smoking with PAH levels found in human milk (Zanieri et al., 2007). These investigators reported individual PAH concentrations in the milk of urban smoking and nonsmoking mothers, and in rural smoking and nonsmoking mothers.

Zanieri et al (2007) had obtained self-reported smoking habits (an arithmetic average of 5.4 cigarettes smoked per day) but not the daily dose of PAHs due to smoking (Zanieri et al., 2007). Therefore, OEHHA estimated daily PAH doses using published estimates of the amounts of PAHs a smoker voluntarily consumes during smoking per cigarette from simulated cigarette smoking studies. (Ding et al., 2005) measured the amount of 14 individual PAHs that would be inhaled because of smoking major U.S. cigarette brands (Table J.3-3). Two other simulated smoking studies were included that estimated the inhaled amounts of two additional PAHs not covered in the Ding study (Gmeiner et al., 1997; Forehand et al., 2000).

Table J.3-3: Summary Estimates of Polycyclic Aromatic Hydrocarbons (PAHs) Intake from Cigarettes (µg/cigarette)

PAH	**Ding et al (n=5)**	**Ding et al (n=50)**	**Ding et al (n=5)**	**Gmeiner et al (n=3)**	**Forehand et al (n=4)**	**Pooled**
With Cancer Potency Factors	**1[#] AM, SD[1]**	**2 AM, SD**	**3 AM, SD**	**1 AM, SD**	**1 AM, SD**	**AM, SD**
Naphthalene	0.3503, 0.021	0.192, 0.044	0.407, 0.187	0.236, 0.019	0.362, 0.011	0.292, 0.087
Chrysene	0.0157, 0.0003	0.0197, 0.0024	0.0314, 0.0028	0.0218, 0.0009	0.0112, 0.0003	0.015, 0.0017
Benzo[a] anthracene	0.0134, 0.0007	0.0165, 0.0015	0.0226, 0.0025	0.0132, 0.0005	0.014, 0.0004	0.015, 0.0014
Benzo[b] fluoranthene	0.0094, 0.003	0.0106, 0.0013	0.0183, 0.0024	0.0086, 0.0003	0.0112, 0.0003	0.010, 0.0012
Benzo[k] fluoranthene	0.0015, 0.00014	0.0019, 0.00029	0.0039, 0.00070	0.0015, 0.00008	NA	0.0020, 0.0004
Benzo[a]pyrene	0.0103, 0.00041	0.011, 0.00077	0.0147, 0.00118	0.0079, 0.00024	0.0076, 0.00023	0.0092, 0.00067
Dibenzo[a,h] anthracene	NA	NA	NA	0.0006, 0.00013	0.0023, 0.00021	0.0023, 0.00017
Indeno[1,2,3-c,d] pyrene	NA	NA	NA	0.0035, 0.00039	NA	0.0035, 0.00039
Without Cancer Potency Factors	1 AM, SD	2 AM, SD	3 AM, SD	1 AM, SD	1 AM, SD	AM, SD
Anthracene	0.0749, 0.0052	0.0698, 0.0084	0.074, 0.0089	0.0381, 0.0023	0.0358, 0.0011	0.043, 0.0060
Acenaphthylene	0.1169, 0.0082	0.0883, 0.0097	0.153, 0.0306	0.0504, 0.0040	NA	0.083, 0.0167
Phenanthrene	0.1348, 0.0054	0.1452, 0.0131	0.144, 0.0144	0.11, 0.0033	0.1477, 0.0044	0.134, 0.0094
Fluorene	0.2175, 0.0087	0.1563, 0.0188	0.257, 0.0257	0.119, 0.0048	0.239, 0.0048	0.184, 0.0151
Acenaphthene	0.0848, 0.0025	0.0513, 0.0072	0.088, 0.0167	0.0253, 0.0013	NA	0.062, 0.0092
Pyrene	0.0486, 0.0029	0.0495, 0.0069	0.077, 0.0231	0.0332, 0.0017	0.0321, 0.0010	0.036, 0.0109
Fluoranthene	0.0744, 0.0037	0.063, 0.0107	0.101, 0.0121	0.0462, 0.0018	0.0516, 0.0026	0.056, 0.0076
Benzo[g,h,i] perylene	NA	NA	NA	0.0025, 0.00030	0.0023, 0.00018	0.0023, 0.00025

[1]AM arithmetic mean,, SD standard deviation ; #, Experiment number listed in the study reference by the first author in row one of columns two through six in the table (Gmeiner et al., 1997; Forehand et al., 2000; Ding et al., 2005)

Based on the estimated intake of 16 measured PAHs in simulated smoking studies and the PAHs found in breast milk from long-time smoking mothers by Zanieri et al. (2007), OEHHA was able to estimate transfer coefficients (Tco) with a modified version of Equation J-1:

$$Tco_{hmi} = Cm_i/(C_{cig\_i} \times I_{cig/day} \times f_{smoke}) \qquad \text{Eq. J-7}$$

where:

Cm_i = adjusted geometric average ith PAH concentration due to smoking (µg per kg milk as wet weight)
C_{cig_i} = geometric average dose of the ith PAH per cigarette (µg/cigarette averaged across experiments)
$I_{cig/day}$ = geometric average number of cigarettes smoked (4.75 cigarettes/day)
f_{smoke} = adjustment for under-reporting of smoking frequency (2)

Cm_i is the adjusted geometric average of the ith PAH in whole milk due to smoking. OEHHA obtained these estimates by converting arithmetic estimates to geometric estimates of the mean and standard deviation and subtracting the GM concentration in the milk of primarily urban nonsmokers from the GM concentration in the milk of urban smokers. This adjustment accounts for oral intake of PAHs from dietary sources and inhalation of PAHs in urban air from combustion sources other than cigarettes. Implicit in this adjustment is the assumption by OEHHA that oral intake and exposure to other airborne PAHs is similar between smokers and nonsmokers who participated in the Zanieri study.

OEHHA also included a 2-fold smoking habit adjustment-factor (f_{smoke}) in Eq. J-7 based on published data to account for the recognized tendency of smokers to under-report their smoking habits. The studies examined the accuracy of self-reported smoking habits among pregnant women and parents with small children (Marbury et al., 1993; Graham and Owen, 2003). They measured airborne nicotine in the smoker's breathing zone and obtained the number of cigarettes smoked per day by each smoker. The data presented in Figure (1) of Marbury et al suggest that mothers under-reported their smoking rate by 50% (Marbury et al., 1993).

Table J.3-4 presents the Tcos for cancer and noncancer PAHs calculated using Eq. J-7. However, Zanieri and Del Bubba did not find measurable levels of some PAHs, particularly PAHs with 5 or 6 carbon rings, in milk from nonsmokers. In these cases, the concentration representing half the limit of detection (between 0.006-0.014 µg/kg) was used as the background concentration of the PAH in mother's milk.

There are two main limitations in the data provided in Table J.3-4. For some PAHs, no individual Tco was calculated because the concentration of the

individual PAH was higher in mother's milk of nonsmokers than in smokers. For example, in column two of Table J.3-4, mother's milk benzo[b]fluoranthene, pyrene and anthracene have negative concentration values.

These discrepancies could be due to the natural variation in the ability of individuals to transfer inhaled PAHs to milk, or as Zaneiri et al. suggested, a result of greater exposure to certain PAHs in some foods compared to cigarette smoke. The small sample sets (n=11 for each group of smokers and nonsmokers) in the Zanieri study are less likely to represent the true mean in the study population and magnify the large variation in this biological response.

Additional uncertainties in the use of smokers to estimate PAH transfer coefficients include that fact that lung metabolism may be different in smokers because of the much higher doses of PAHs that smokers receive relative to those only exposed in ambient pollution. Cytochrome P-450 enzymes are known to be induced when exposure is greater and therefore metabolism could be proportionately greater in smokers. In addition, at higher dose levels some enzyme systems may become saturated which could alter the pattern of metabolism.

However, smokers are the best population for estimating PAH Tcos because the inhalation dose can be separated from background inhalation and dietary exposure, and the inhalation dose from the cigarettes can be estimated. OEHHA requested raw data from the investigators for individual women in the study, but unfortunately, only the summary statistics from the published paper were available to us.

Table J.3-4: Inhalation Transfer Coefficients (Tcos) for Individual PAHs with and without Potency factors from Geometric Mean and Standard Deviation Estimates (GM, GSD) of Human Milk (Cm) and Intake from Cigarettes (Ccig) (d/kg-milk)

PAH (no. of rings)[a]	Adjusted Cm (µg/kg wet wt.)	C_{cig} (µg/cig)	Inhalation Tco[b] (d/kg)
With Cancer Potency Factors	**GM, GSD**	**GM, GSD**	**GM, GSD**
Naphthalene (2)	2.78, 1.63	0.2798, 1.34	1, 2.66
Chrysene (4)	0.04, 5.34	0.0149, 1.12	0.28, 8.11
Benzo[a]anthracene (4)	0.20, 4.31	0.0149, 1.1	1.4, 6.52
Benzo[b]fluoranthene (5)	*-0.09, 5.01*	0.0099, 1.13	NA[c]
Benzo[k]fluoranthene (5)	0.05, 2.95	0.002, 1.22	0.26, 4.6
Benzo[a]pyrene (5)	0.26, 2.29	0.0092, 1.08	2.97, 3.45
Dibenzo[a,h]anthracene (5)	0.46, 3.85	0.0023, 1.08	2.11, 5.81
Indeno[1,2,3-c,d]pyrene (6)	0.16, 3.65	0.0035, 1.12	4.81, 5.54
Without Cancer Potency Factors	**GM, GSD**	**GM, GSD**	**GM, GSD**
Anthracene (3)	*-0.22, 6.29*	0.0426, 1.15	NA
Acenaphthylene (3)	*-4.56, 2.9*	0.0814, 1.22	NA
Phenanthrene (3)	2.00, 1.94	0.0035, 1.07	1.57, 2.92
Fluorene (3)	1.31, 4.1	0.1336, 1.09	0.75, 6.19
Acenaphthene (3)	2.48, 3.26	0.0613, 1.16	4.21, 5
Pyrene (4)	0.04, 4.57	0.0345, 1.34	0.12, 7.48
Fluoranthene (4)	1.63, 3.29	0.0555, 1.14	3.06, 5.02
Benzo[g,h,i]perylene (6)	0.77, 2.72	0.0023, 1.11	35.24, 4.13

[a] no. of rings, number of rings are an indicator of lipophilicity (greater # of rings, more likely to partition to body fat); [b] Sum of each PAH found in mother's milk microgram per kilogram (µg/kg) over the sum of the daily intake (µg/day) of the same PAH x 4.75 cigarettes/day x an adjustment factor of 2; [c] NA, not available because the concentration of PAH in mother's milk of smokers was lower than the concentration in nonsmokers, so an individual Tco could be calculated

Tco values for carcinogenic PAHs in Table J.3-4 are determined for all available PAHs and included in a summary estimate (see Table J.3-7 near the end of this section).

Unlike the other PAHs with cancer potency factors, naphthalene is not considered a multipathway chemical under the Hot Spots program because it is regarded as a gas, and therefore not subject to appreciable deposition onto soil,

etc. Naphthalene was included in this analysis because this PAH constitutes a large proportion of the total mass of PAHs inhaled. Among the carcinogenic PAHs in Table J.3-4, naphthalene predominates in both mainstream smoke (63% of total carcinogenic PAHs) and in mother's milk (56% of total carcinogenic PAHs). Naphthalene is also the only PAH that is considered a gas, and therefore, its physical properties are different from other larger PAHs that are semi-volatile or exist primarily as a solid. In spite of these differences, the summary estimate did not change when naphthalene was excluded in the analysis (summary Tco = 1.55 versus 1.60).

Due to few measurable levels of carcinogenic PAHs in milk samples, there is more uncertainty in the carcinogenic PAH Tco compared to the PAH Tco for PAHs without cancer potency values. Nevertheless, summary estimates for PAH Tcos from inhaled sources differ by less than a factor of two (Tco for carcinogens, 1.2 versus Tco without cancer potency values, 2.06) suggesting that there may be no systematic difference between these two groups of chemicals. Therefore, OEHHA combined individual Tcos for PAHs from both groups into an overall inhalation Tco (see Table J.3-7 and Figure J.3-1 at the end of this section of the Appendix). In Figure J.3-1, the top seven estimates of inhalation Tcos are carcinogenic PAHs and the bottom six estimates are PAHs without cancer potency values.

The combined estimate is the summary of all 13 PAH estimates combined using a Random-effects model. OEHHA assumes that the PAHs found in exposure studies are a subgroup from a population of PAHs. Random-effects models assume there are multiple central estimates and incorporate a between-PAH estimate of error as well as a within-PAH estimate of error. In contrast, a Fixed-effects model assumes observations scatter about one central estimate (Kleinbaum, 1988).

OEHHA recommends using the inhalation Tco based on the summary estimates provided in Table J.3-7 rather than using the individual PAH Tcos values provided in Table J.3-4, to assess transfer of individual inhaled PAHs to mother's milk. There are a high number of non-detects and small sample sizes in these data. The estimation of PAH Tco values with this method might be improved with more sensitive methods for measurement of breast milk PAH content and larger study populations to better estimate biological variation and estimates of PAH transfer from air to mother's milk. Such improved data could allow for a robust determination of the Tco values for individual compounds.

The key assumption underlying the development of these Tcos is that the variability in individual PAHs Tcos is sufficiently small to justify the use of an average value for individual PAH congeners. This approach appears to be the best available given the available studies.

J.3.2 Oral Biotransfer of PAHs to Mother's Milk

Diet is the largest contributor by pathway to total PAH intake from ubiquitous background sources for the general public and other situations where airborne levels are not remarkably high (Lioy et al., 1988). In a risk assessment of a reference nonsmoking male, a mean total PAH intake of 3.12 µg/d was estimated of which dietary intake was 96.2%, air 1.6%, water 0.2% and soil 0.4% (Menzie et al., 1992; Ramesh et al., 2004). Inhalation, soil ingestion and homegrown produce pathways can be important when considering total dose from a single stationary source. PAHs contaminate homegrown produce and soil through direct deposition. Milk and meat from home-raised animals or commercial sources would be less of a contributor because many PAHs are highly metabolized by these animals following intake from contaminated pastures and soil.

There are no studies available that relate PAH dietary intake directly to mother's milk concentrations for these compounds, although studies of PAH dietary intake have been performed in several countries. Therefore, the PAH biotransfer efficiency to mother's milk from food was calculated using PAH dietary intake data and mother's milk PAH data from separate studies. OEHHA recognizes the uncertainty in this approach but it appears to be the best currently available. Table J.3-5 shows the daily dietary intake of carcinogenic PAHs from published studies of European residents.

Regional preferences, ethnicity, and individual dietary preferences will influence the amount of PAHs ingested with food. In addition, there were differences among the intake studies in the number and type of PAHs investigated in foods. Even though dietary habits and PAH analysis methods can result in different levels of PAH intake, the total dietary intakes of PAHs in each of five studies in Table J.3-5 were generally within an order of magnitude of each other.

Table J.3-5: Summary Estimates of PAHs with and without Cancer Potency Factors Dietary Intake (µg/day)

PAH (no. of rings[a])	Italian Lodovici et al (1995) Adults	Dutch De Vos et al. (1990) [c] Adult males	Spanish Marti-Cid et al. (2008) Adults	Spanish Falco et al. (2003) Adults	U.K. Dennis et al. (1983) Adults
With Cancer Potency Factors	**AM[b], SD**	**AM***	**AM***	**AM, SD**	**AM***
Naphthalene (2)	NA[d]	NA	1.846	0.823, 0.056	NA
Chrysene (4)	0.84, 0.0131	0.86 – 1.53	0.204	0.564, 0.037	0.5
Benzo[a]anthracene (4)	0.47, 0.0093	0.2 – 0.36	0.139	0.310, 0.021	0.22
Benzo[b]fluoranthene (5)	0.17, 0.0101	0.31 – 0.36	0.137	0.188, 0.014	0.18
Benzo[k]fluoranthene (5)	0.06, 0.0043	0.1 – 0.14	0.086	0.094, 0.006	0.06
Benzo[a]pyrene (5)	0.13, 0.0003	0.12 – 0.29	0.083	0.113, 0.008	0.25
Dibenzo[a,h]anthracene (5)	0.01, 0.0026	ND[e]	0.084	0.048, 0.003	0.03
Indeno[1,2,3-c,d]pyrene (6)	ND	0.08 – 0.46	0.102	0.045, 0.003	ND
Without Cancer Potency Factors	**AM, SD**	**AM***	**AM***	**AM, SD**	**AM***
Anthracene (3)	NA	0.03 – 0.64	0.428	0.088, 0.006	NA
Acenaphthylene (3)	NA	NA	0.354	0.402, 0.026	NA
Phenanthrene (3)	NA	NA	3.568	2.062, 0.150	NA
Fluorene (3)	NA	NA	0.934	0.206, 0.017	NA
Acenaphthene (3)	NA	NA	0.368	0.071, 0.005	NA
Pyrene (4)	0.19, 0.0043	NA	1.084	1.273, 0.092	1.09
Fluoranthene (4)	1.03, 0.0106	0.99 – 1.66	1.446	0.848, 0.062	0.99
Benzo[g,h,i]perylene (6)	0.20, 0.0009	0.2 – 0.36	0.112	0.214, 0.017	0.21

[a] no. of rings, number of rings are an indicator of lipophilicity (greater # of rings, more likely to partition to body fat); [b] Arithmetic mean (AM), Standard Deviation (SD); [c] The Dutch dietary intakes were presented as the range of lower bound values (calculated by taking values below the detection limit to be zero) to upper bound values (calculated by taking values below the detection limit to be equal to the limit) [d] *NA,* Not available; [e] ND, Not determined; * no measure of variance was reported (Dennis et al., 1983a; Dennis et al., 1983b; De Vos et al., 1990; Lodovici et al., 1995; Falcó et al., 2003; Martí-Cid et al., 2008)

Mother's milk PAH concentration data from nonsmoking rural lactating women living in Italy by Zaneiri et al. (2007) and Del Bubba et al. (2005) were pooled and paired with estimates of PAH dietary intake in the Italian population (Lodovici et al., 1995; Del Bubba et al., 2005; Zanieri et al., 2007). The mother's milk PAH data are described above in the PAH inhalation biotransfer section. The study by

Del Bubba et al. is a similar study by the same research group that includes additional participants from rural areas. The use of nonsmoking rural women should reduce confounding contributions from the inhalation pathway. Airborne concentrations of PAHs tend to be higher in urban areas due to mobile sources.

Based on the estimated intake of the same measured PAHs in dietary studies and the PAHs found in breast milk from nonsmoking mothers (Del Bubba et al., 2005; Zanieri et al., 2007), OEHHA was able to estimate transfer coefficients (Tco) by Equation J-8, a version of Equation J-1:

$Tco_{hmoi} = Cm_{oi} /(D_{oi})$ Eq. J-8

where:

Cm_{oi} = geometric average ith PAH concentration in mother's milk (µg per kg milk as wet weight)
D_{oi} = geometric average dose of the ith PAH per day from dietary sources (µg/day)

Cm_{oi} is the geometric average of the ith PAH in whole milk from nonsmoking, rural dwelling women. OEHHA obtained estimates of GM and GSD by pooling and converting arithmetic estimates to geometric estimates of the mean and standard deviation from two studies of nonsmoking rural-dwelling women (Del Bubba et al., 2005; Zanieri et al., 2007). D_{oi} is the geometric average of the ith PAH taken in through dietary sources. Oral PAH Tcos for both carcinogenic and noncancer PAHs are shown in Table J.3-6.

The Italian dietary study by Lodovici et al. (1995) supplied data in which OEHHA could calculate estimates of dietary intake of nine PAHs among a population living mostly in urban settings. OEHHA obtained GM and GSD estimates by converting arithmetic estimates of dietary intake reported in Lodovici et al (1995) and estimates of intake variability from Buiatti et al (1989).

These investigators estimated that the entire study population consumes about 1.9 µg of carcinogenic PAHs per day from dietary sources. Approximately 46% of the total carcinogenic PAH intake comes from cereal products, non-barbecued meat, oils and fats. Even though meat barbecued on wood charcoal has the highest PAH levels, the contribution of these barbecued foods is only about 13% of the carcinogenic PAH intake.

A limitation of the Italian dietary intake study is that the population examined was 58% men, and the study did not report any body weight adjustments. Thus, the sample population may not represent the female population sampled by Zanieri et al (2007). Other studies that have compared dietary PAH intake levels between men and women indicate that men consume slightly higher levels of

PAHs than women do (5% to 15% on a µg/kg-body weight-day basis) (Falco et al 2003, Marti-Cid et al 2008), so the bias introduced by this assumption may not be significant.

Table J.3-6 presents the dietary intake and mother's milk concentrations for individual PAHs from the Italian studies. OEHHA calculated Tcos for individual PAHs common to both the studies of dietary intake and mother's milk concentration. The mother's milk concentrations for individual PAHs represents the pooled average reported in the Zanieri et al. and Del Bubba et al. studies.

Table J.3-6: Oral Transfer Coefficients (Tcos) for Individual PAHs Based on Italian Data from a Daily PAH Dietary Intake Study (Lodovici et al., 1995; Del Bubba et al., 2005; Zanieri et al., 2007) and Mother's Milk PAH Concentration Studies (Del Bubba et al., 2005; Zanieri et al., 2007).

PAH	Mother's milk PAH concentration (µg/kg-milk)	Daily PAH intake (µg/d)	Oral PAH Tco (d/kg)
With Cancer Potency Factors	GM[a], GSD[b]	GM, GSD	GM, GSD
Naphthalene	4.12, 1.41	NA[c]	NA
Chrysene	0.01, 3.36	0.49, 2.82	0.02, 4.93
Benzo[a]anthracene	0.12, 5.41	0.27, 2.82	0.44, 7.25
Benzo[b]fluoranthene	0.21, 3.61	0.1, 2.82	2.1, 5.21
Benzo[k]fluoranthene	0.055, 3.01	0.034, 2.82	1.62, 4.54
Benzo[a]pyrene	0.01, 3.36	0.076, 2.82	0.13, 4.93
Dibenzo[a,h]anthracene	0.007, 3.36	0.003, 2.82	2.33, 4.93
Indeno[1,2,3-c,d]pyrene	0.011, 3.36	NA	NA
Without Cancer Potency Factors	GM, GSD	GM, GSD	GM, GSD
Anthracene	0.13, 4.26	NA	NA
Acenaphthylene	4, 1.99	NA	NA
Phenanthrene	0.41, 2.03	NA	NA
Fluorene	0.12, 6.32	NA	NA
Acenaphthene	1.39, 2.16	NA	NA
Pyrene	0.15, 3.47	0.11, 2.82	1.35, 5.05
Fluoranthene	0.16, 3.34	0.6, 2.82	0.27, 4.91
Benzo[g,h,i]perylene	0.01, 3.37	0.116, 2.82	0.08, 4.94

[a] GM, geometric mean; [b] GSD, geometric standard deviation; [c] NA, Not available;

Oral Tcos were calculated for each individual PAH by equation J-8. The average Tco for carcinogenic and PAHs without cancer potency factors was calculated as the sum of the Tco values over the total number of PAHs evaluated. Similar Tco values are obtained for both groups of PAHs (0.46 d/kg) and 0.31 d/kg, respectively). This finding suggests that, on average, the PAHs with cancer potency factors as a whole transfer to mother's milk with about the same

efficiency as some of the most common PAHs without cancer potency factors that are taken in through the diet.

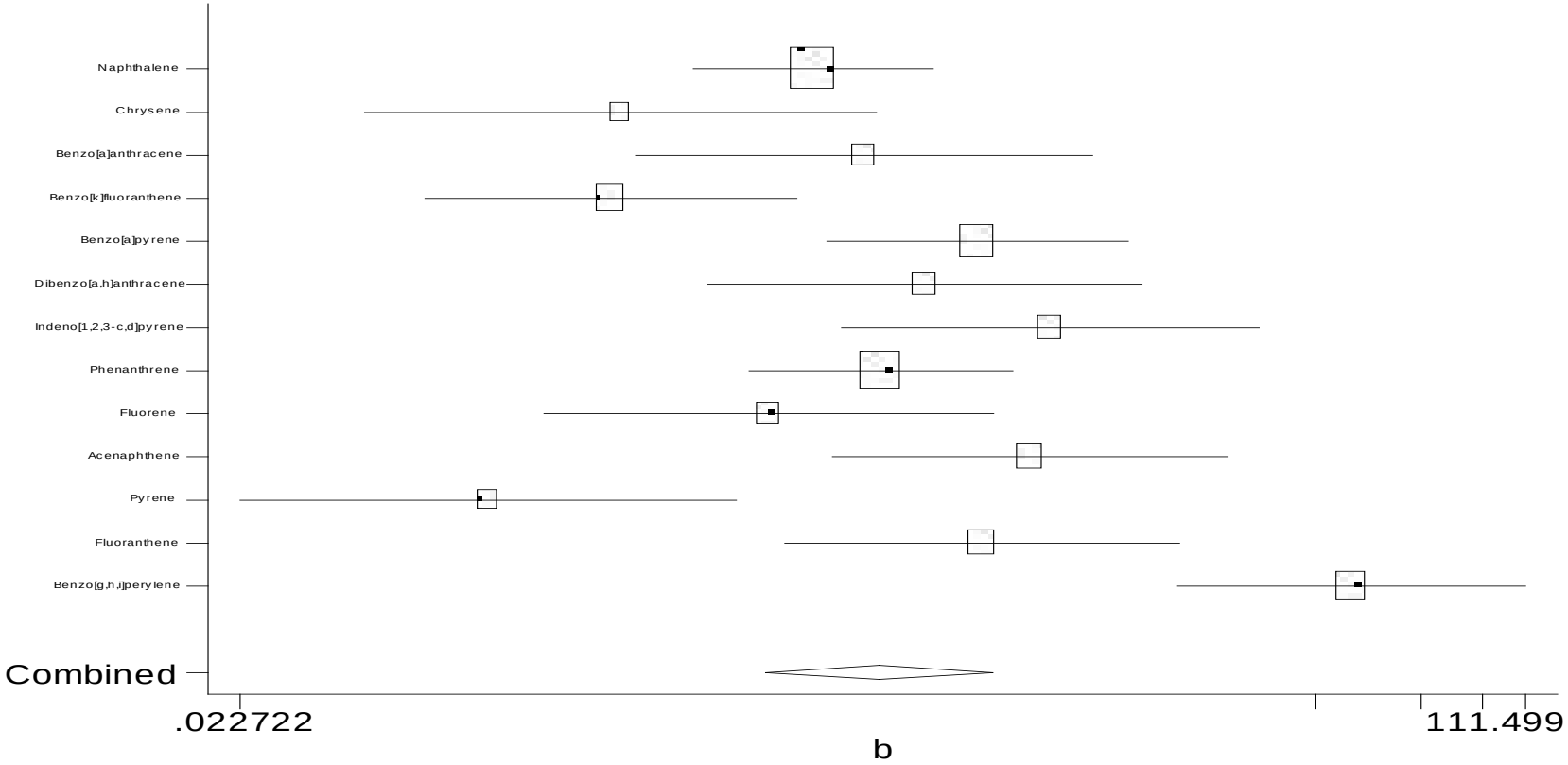
Summary Tcos were calculated using a Random-effects model to pool across individual PAH-Tcos. OEHHA found no systematic difference between summary estimates stratified by PAHs with or without cancer potency factors (data not shown). Therefore, we pooled Tcos for both groups by route of intake (see Table J.3-7).

Table J.3-7: Random Effects Estimate and 95% Confidence Intervals of Tcos Stratified by Intake Route and Data Source

Tco (data source)	No. PAHs	summary estimate (random effects model)	LCL	UCL
Inhalation	13	1.55	0.731	3.281
Oral (Italian)	9	0.401	0.132	1.218

LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco.

Figure J.3-1: Inhalation Tcos (b, 95% CL) Based on Italian Data, (Random-effects Model)



Top seven estimates are PAHs with potency factors and bottom six estimates are PAHs without potency factors; summary of all 13 PAHs is labeled “combined” = 1.55 d/kg; b, the Tco in units of day/kg-milk

Similar to the inhalation Tco derivation, limitations of the oral Tco derivations include the small number of women examined for PAHs in mother's milk (n=21) and the large number of "below detection limit" results for milk concentrations, particularly for the larger PAHs with more than four rings. OEHHA assumed that the arithmetic estimates, minimum and maximum values reported by investigators represented a lognormal distribution and converted estimates from arithmetic to geometric. Nevertheless, the use of sparse data to derive an inhalation Tco and data from potentially two different study populations to generate an oral Tco – one for dietary PAH intake and another for mother's milk PAH concentrations - introduces considerable uncertainty.

J.3.3 Comparison and Use of Inhalation and Oral PAH Tcos

Comparison of the oral and inhalation Tcos also presents a number of interesting findings. For example, comparing the averaged inhalation and oral mother's milk Tcos generated from the Italian studies for carcinogenic PAHs, the mean inhalation Tco is about four times greater than the oral Tcos based on Italian study data.

Although studies in humans are lacking, (Grova et al., 2002) showed that BaP is poorly absorbed through the gut in goats when administered orally in vegetable oil. Radiolabeled BaP fed to these animals led to 88% recovery of the radioactivity in feces, indicating little BaP reached the bloodstream where it could be taken up in mother's milk. In contrast, respiratory absorption of PAHs in particulate form through smoking is about 75% efficient (Van Rooij et al., 1994).

The following factors may have influenced the difference between oral Tco values and inhalation Tco values:

- First-pass metabolism in the liver following oral intake before reaching the blood supply of the breast versus entering systemic blood circulation prior to passage through the liver with the inhalation route (however, some PAH metabolism occurs in the lung)
- Gut assimilation of PAHs is likely to occur at a different rate than the rate of passage across the lung

Looking at mother's milk Tcos in terms of carryover rate suggests that accumulation of PAHs in the mother's body occurs more readily when inhaled versus ingested. Carryover rate, defined here as the daily output of PAHs in mother's milk (µg/day) over the daily intake of PAHs (µg/day), can be estimated by multiplying a PAH Tco by the daily output of mother's milk. Since milk production in human mothers are about 1.0 kg/day, the calculated carryover rate turns out to be the same as the PAH Tco value. A carryover rate greater than one in PAH transfer suggests that accumulation occurs in the mother's body prior to lactation.

The average inhalation Tco of 1.6 d/kg daily inhalation of a PAH mixture, indicates that 160% of the daily intake from inhaled sources transfers to mother's milk. This high transfer-value suggests that some accumulation of PAHs with cancer potency factors may occur in the mother's body before lactation begins. An average oral Tco of 0.40 d/kg for PAHs with cancer potency factors indicates 40% of the daily intake from diet transfers to mother's milk following oral intake of PAHs.

This suggests that metabolism occurs in the mother's body. The uncertainties in our Tco estimation methods could account for both of these results. If the Tco estimation is correct, the mother may be metabolizing a considerable fraction of her intake prior to partitioning into the fat stores. There could also be inefficient transfer to mother's milk for unknown reasons or metabolism following transfer of PAHs to mother's milk.

J.4 Mothers' Milk Transfer Coefficients for Inorganic Lead

Inorganic lead is naturally present on the earth's crust and may enter terrestrial and aquatic ecosystems due to the weathering of rocks. Traces of lead can not only be found in the immediate vicinity of emission sources but also are present, albeit at very low levels, in every part of the world (Castellino and Castellino, 1995).

Lead particulate matter is the primary form of lead present in the air (OEHHA 1997). Atmospheric movements may transport lead aerosol in the form of very fine particles, a long way from its place of emission. Refineries, mineral extraction industries, and smelting plants for lead and other metals are largely responsible for emitting lead-containing aerosols into the atmosphere (Castellino and Castellino, 1995) in the U.S.

Human intake of lead can occur by inhalation of airborne particles and ingestion of lead-contaminated food and water. Furthermore, people can be exposed using lead-glazed or painted cooking and eating utensils. Lead may also be ingested in foods or drinks contaminated with the metal during the industrial processes of food production or preservation (Castellino and Castellino, 1995). The potential pathways of concern with Hot Spots facilities would be inhalation, soil ingestion, and dermal absorption, home raised meat, homegrown produce, surface drinking water consumption, and breast milk consumption.

Background levels of lead in the blood of the U.S. population have declined in recent years mainly resulting from the removal of lead from gasoline and paint. Results from an NHANES study (1991 – 1994) show that the geometric mean blood lead level in the U.S. adult population (20 – 69 years of age) was about 4 µg/dL (Pirkle et al., 1994), which is over a 70% decline in blood lead from blood lead levels obtained from 1976 to 1980. The NHANES IV survey (1999- 2000)

found an additional 50% reduction (1.75 µg/dL) in the U.S. adult population (CDC, 2005).

As of the date of this report, measured levels of lead at ambient air quality monitoring sites in California are very low. Lead exposure in the California population is likely to occur from sources other than Hot Spots facility emissions, such as old lead-based paint. However, no threshold has been identified for lead-induced neurotoxicity in children and therefore an evaluation of all potential routes of exposure for Hot Spots facilities is prudent. Further, there are significant lead emissions from some Hot Spots facilities.

In an effort to derive lactation transfer coefficients for inorganic lead, OEHHA drew from studies conducted on subjects exposed to lead through multiple pathways at higher levels from other areas of the world. OEHHA assumes that the transfer of lead derived from these studies serves as a reasonable surrogate for the transfer of lead from contaminated media near a Hot Spots facility in California.

J.4.1 Inorganic Lead in Human Milk

Breast milk levels of lead correlate with levels of lead in whole blood but are generally much lower (Sternowsky and Wessolowski, 1985; Castellino and Castellino, 1995; Li et al., 2000; Ettinger et al., 2004). Castellino et al (1995) reviewed 11 studies conducted between 1933 to 1989 and observed that in the vast majority of cases, the mean values of lead in breast milk vary from 0.17 to 5.6 µg/L (Castellino and Castellino, 1995).

Ursinyova and Masamova (2005) published a table of 32 human milk summary estimates from studies published between 1983 and 2001. Mean human milk levels of lead generally ranged from 0.5 to 50 µg/L (Ursinyova and Masanova, 2005). Average blood lead levels during that timeframe ranged from 24 to 460 (µg/L) (Gulson et al., 1998a).

Because lead levels in milk correlate well with whole blood, OEHHA searched for studies that reported both lead levels in milk and blood before and/or during lactation for derivation of a lactational Tco for lead. However, several investigators have questioned high results from early studies of lead in breast milk. For example, Ettinger et al (2004), Gulson (1998b) and others cautioned that high levels of lead in breast milk might be due to contamination from some past sample collection techniques (Hu et al., 1996; Newman, 1997; Gulson et al., 1998a; Smith et al., 1998; Ettinger et al., 2004). These sources of lead include the use of the following products to prepare nipples or express breast milk:

- lead acetate ointment
- lead in nipple shields
- lead in alcohol wipes from foil wrap

Gulson et al (1998a) also suggested that analytical problems, indicated by an unusually wide range in lead concentrations for the quality control standard in Parr et al (1991), warrant verification by follow-up studies (Parr et al., 1991; Gulson et al., 1998a). Gulson et al (1998a) assessed lead concentrations in maternal blood versus the concentration of lead in breast milk per concentration in maternal whole blood from studies conducted over 15 years prior to 1998. From this assessment, they suggested that milk lead levels less than about 15% of maternal blood lead levels best represent the relationship between lead in maternal blood and milk. In other words, milk lead levels that were greater than 15% of blood lead levels were suspected of being contaminated with lead during sample collection and/or assessment. Therefore, OEHHA has included only summary estimates from studies published after 1990 that did not report or show evidence of breast milk contamination.

OEHHA located eight studies that met our inclusion criteria. Table J.4-1 summarizes key attributes of the study populations.

Table J.4-1: Studies with Summary Estimates of Concurrent Maternal Blood and Milk Levels of Lead)

Study	Country	Group	Study period	Measurement	# Study subjects
(Nashashibi et al., 1999)	Greece	Residents of Athens and surrounding areas	~1999	At delivery, at onset of lactation	47
(Li et al., 2000)	China, Shanghai	Not occupationally exposed	prior to 2000	At delivery, at onset of lactation	32
(Counter et al., 2004)	Equador, Pujili	Pottery glazers	2003	Post partum	13
(Ettinger et al., 2004)	Mexico, Mexico City	Exclusive breast feeders	1994-1995	One month postpartum	88
(Ettinger et al., 2004)	Mexico, Mexico City	Partial breast feeders	1994-1995	One month postpartum	165
(Namihira et al., 1993)	Mexico (Mexico City)	Reside near New Smelter	1986	postpartum	35
(Hallen et al., 1995)	Sweden	Reside in Rural areas	1990-1992	6 weeks postpartum	39
(Hallen et al., 1995)	Sweden	Reside near Smelter area	1990-1992	6 weeks postpartum	35
(Baum and Shannon, 1996)	U.S.A Camden, New Jersey	Mothers of lead poisoned infants	1996	Postpartum	2
(Gulson et al., 1998b)	Australia	Immigrants from eastern Europe	Early 1990s	At delivery and average during lactation	9

Regression analyses suggest a linear relationship between lead in maternal blood and milk among women with substantially elevated levels of lead in blood. For example, Namihira et al (1993) reported a significant linear relationship (r = 0.88) between levels of lead in blood and milk for blood lead levels in the range of 35 µg/dL -100 µg/dL from a study of 35 lactating women living in Mexico City (Namihira et al., 1993). At these levels of lead in blood, authors reported a univariate regression of 4.3% representing the average level of lead in breast milk relative to the average level of lead in blood.

A similar study of 47 lactating women conducted by Nashashibi et al also reported a significant linear relationship (r=0.77) between lead in milk and blood for blood lead levels in the range of 5 µg/dL - 25 µg/dL (Nashashibi et al., 1999).

Based on a univariate regression, the average level of lead in breast milk was about 7% the average level of lead in blood. OEHHA calculated similar estimates of the milk/blood lead ratio from Li et al (2000), Counter et al (2002) and Ettinger et al (2004) (see Table J.4-2).

Table J.4-2 Concurrent Measurements of the Lead Concentration (µg/L) in Mother's Milk and Blood

Study	N	Blood AM,SD	Milk AM,SD	Blood GM,GSD	Milk GM,GSD
(Nashashibi et al., 1999)	47	149, 41.1	20,5	143.64, 1.31	19.4, 1.28
(Li et al., 2000)	119	142.5, 69.14	5.63,4.39	128.21, 1.58	4.44, 1.99
(Counter et al., 2004)	13	171, 91	4.6,5.3	150.96, 1.65	3.02, 2.51
(Ettinger et al., 2004)	88[a]	94, 48	1.4,1.1	83.72, 1.62	1.1, 2
(Ettinger et al., 2004)	165[b]	95, 43	1.5,1.2	86.55, 1.54	1.17, 2.02
(Namihira et al., 1993)	35	459, 198.8	29.94,25.75	421.19, 1.51	24.7, 1.86
(Hallen et al., 1995)	39[c]	31.4, 6.7	0.5,0.3**	30.71, 1.23	0.43, 1.74
(Hallen et al., 1995)	35[d]	31.7, 10.2	0.9,0.4***	30.18, 1.37	0.82, 1.53
(Baum and Shannon, 1996)	2	315, 35.4	5.02,0.50	313.03, 1.12	5, 1.1
(Gulson et al., 1998b)	9	29, 8	0.73,0.7	27.96, 1.31	0.53, 2.24

[a]exclusively breast fed; [b] partially breast fed; [c] rural setting; [d] near smelter; * < LOD taken as 1/2 LOD as GM and 9.9 = max, **based on LOD of 0.5 µg/L and 2 out of 39 samples above LOD; *** based on 16/35 above LOD

Li et al. (2000) stratified milk lead levels by low, medium and high blood lead levels. Their findings suggest that slightly higher transfer rates occur at low levels relative to high levels of lead in blood (Li et al., 2000). This may be due to more efficient transfer rates at lower body burdens of lead or it could result from very slight breast milk contamination during collection and/or assessment.

J.4.2 Biotransfer from Bone to Blood during Pregnancy and Lactation

Lead transferred from blood to human milk reflects both the mother's current and ongoing intake of lead exposure as well as lead mobilized due to physiological changes of pregnancy and lactation from bone stores due to past exposures. Several studies provided indications of internal transfer of lead from bone stores. Internal transfer was evident by comparing the rise in blood lead levels during lactation to blood lead levels measured prior to lactation (see Table J.4-3).

Table J.4-3: Change in Blood Lead Levels from Pregnancy (bloodpreg) to Lactation (bloodlac) (µg/L)

Study		Bloodpreg	Bloodlac	Bloodpreg	Bloodlac
	N	AM,SD	AM,SD	GM,GSD	GM,GSD
(Gulson et al., 1997)**	8	22.4, 6	32, 8.4	21.64, 1.30	30.95, 1.29
(Ettinger et al., 2004)	~86-88excl	81, 38	94, 48	73.33, 1.56	83.72, 1.62
(Ettinger et al., 2004)	164-165part	90, 44	95, 43	80.85, 1.59	86.55, 1.54
(Tellez-Rojo et al., 2002)	425	84, 40	93.7, 43.04	75.84, 1.57	85.15, 1.55
(Sowers et al., 2002)*	15	13.7, 7.75	17, 5.29	11.93, 1.69	16.23, 1.36
(Rothenberg et al., 2000)	311	27.59, 26.49	32.03, 21.78	22, 1.96	28, 1.68

* SD for blood lead level during lactation estimated for blood lead at 6-months from figure 2; ** bloodlact is max blood lead level during pregnancy and lactation; excl, exclusively breastfed; part, partially breastfed

These investigators conducted longitudinal monitoring of blood samples to determine stable lead isotope profiles by mass spectrometry and chemical analyses of blood samples for total lead content over a 300-day period. Gulson et al followed Australian women (15 immigrants and 7 non-immigrants) to study the mobilization of lead from the maternal skeleton during pregnancy and lactation (Gulson et al., 1995; Gulson et al., 1997; Gulson et al., 1998a; Gulson et al., 1998b; Gulson et al., 1999; Gulson et al., 2001). Investigators measured maternal and infant blood, urine, diet, and breast milk from 21 mothers and 24 infants. The arithmetic mean and standard deviation lead concentration in breast milk were AM (SD) 0.73 (0.70) µg/kg and the geometric mean and standard deviation were GM (GSD) 0.55 (2.24) respectively. Levels ranged from 0.09 to 3.1 µg/kg.

Gulson et al (1997) provided evidence that lead in female immigrants to Australia was mobilized from skeletal stores during pregnancy, with increases in blood lead concentration of about 20% and a mean increase in skeletal lead contribution to blood lead of 31%. Authors concluded that between 45% and 70% of lead in blood comes from mobilized long-term tissue lead stores (Gulson et al., 1997).

Investigators obtained environmental samples of house dust, drinking water, urban air, gasoline, and a 6-day duplicate diet quarterly. The GM (GSD) blood lead concentration for the immigrant females on arrival in Australia (either prior to or during early pregnancy) was 3.0 µg/dL (SD 1.56) (range: 1.9 to 20 µg/dL) and for the Australian controls was 3.1 µg/dL (range: 1.9 to 4.3 µg/dL). Skeletal lead

contribution to blood lead was significantly greater ($p < 0.001$) during the post pregnancy period than during the 2nd and 3rd trimesters.

The contribution of skeletal lead to blood lead during the post-pregnancy period remained constant at the increased level even though the duration of breast-feeding varied from 1 week to 6 months. The authors concluded that the increased contribution of skeletal lead both during pregnancy and in the post pregnancy period is consistent with increased bone resorption and may be associated with inadequate calcium intake.

Sowers et al (2000) followed lactating women enrolled in prenatal program located in Camden, New Jersey between 1997 and 2000 (Sowers et al., 2002). These women were part of a larger cohort of 962 women enrolled in study of calcium metabolism in pregnancy and lactation. A nested cohort of 15 women with a mean (standard deviation) age of 23.7 (5.42) years, who provided breast milk samples through 6 months postpartum or longer and were unaware of their blood lead levels, was included in the study. Blood and milk lead levels along with measures of bone loss and osteocalcin concentrations were evaluated. Authors reported the precautions taken to avoid contamination of milk samples by environmental lead.

The arithmetic mean (standard deviation) (µg/dL) of blood lead levels at delivery for 15 breast-feeding and 30 randomly selected bottle-feeding women were 1.37 (0.78) and 1.31 (1.10) respectively. Mean maternal blood lead levels rose to 1.6, (1.7) µg/dL at three and six months during lactation, respectively. Compared to bottle-feeding women, blood lead levels from breast-feeding women were consistently higher by 15 – 35% during the first six months postpartum. Authors found that breast-feeding women had greater bone loss as reflected in the bone change data and higher serum osteocalcin concentrations than bottle-feeding women.

The arithmetic mean of lead in breast milk samples (standard deviation) were 5.6 (4.2) and 5.9 (3.87) µg/L at three and six months post partum. Breast milk lead was also measured 1.5 and 12 months post partum. However, authors did not measure blood lead at 1.5 months, did not indicate how many women were still breast-feeding and did not attempt to estimate how many liters/day study subjects produced. The relative increase in blood lead levels from delivery to an active lactating period (e.g. one to 6 months) is consistent with the relative increases in blood lead found in other studies (see Table J.4-3).

Tellez-Rojo et al (2002) concluded that maternal bone lead levels are an important predictor of maternal blood lead levels over the course of lactation. In fact, bone lead from past exposures can contribute an additional 40% of the lead measured in blood during lactation (see Table J.4-3) (Tellez-Rojo et al., 2002).

Ettinger et al (2004) measured relatively high maternal blood lead levels in women exposed to lead in the air while living in Mexico City. Between January 1994 and June 1995, investigators selected 1398 women from three maternity hospitals in Mexico City for participation in a randomized control trial (Tellez-Rojo et al., 2002; Hernandez-Avila et al., 2003; Ettinger et al., 2004). From this study population, 629 women agreed to participate. Ettinger et al. (2004) examined a nested cohort of 255 women with a mean (standard deviation) age of 24 (5) years with both breast milk, maternal and infant blood lead levels at delivery and one-month post partum. The authors reported the precautions taken to avoid contamination of milk samples by environmental lead.

For breast-feeding women, the arithmetic mean (standard deviation) of blood lead level at delivery was 8.7 (4.2) and at one-month post partum was 9.4 (4.5) µg/dL. At one-month post partum, the average (standard deviation) lead level in breast milk was 1.5 (1.2) µg/L. After adjusting for parity, calcium intake, infant weight change and breastfeeding status, an increase in blood lead was associated with a 33% increase in breast milk lead.

Rothenberg et al (2000) recruited immigrant women, almost exclusively from Latin America, from outpatient clinics in South Central Los Angeles to examine bone lead contribution to blood lead. Investigators contacted subjects from June 1995 through July 1998. Three hundred eleven subjects were followed from late pregnancy to one or two months after delivery. The investigators evaluated bone lead levels after delivery and blood lead levels both pre- and post-delivery. Ages ranged from 15 to 44 years. Prenatal blood lead was lower on average GM = 2.2 µg/dL (0.4 to 38.7) than postnatal blood lead GM = 2.8 µg/dL (0.4 to 25.4). In fact, postnatal blood lead level increased by 27% relative to the prenatal blood lead level.

A questionnaire was administered including questions about present breast feeding practice (presently nursing yes/no) and past history of breast feeding (ever nursed and total months nursed). Breast milk samples were not obtained from this cohort. Tibia and calcaneus bone lead levels were associated with prenatal blood lead levels and calcaneus but not tibia lead was associated with postnatal blood lead levels (Rothenberg et al., 2000).

J.4.3 Inhalation Biotransfer of Lead to Mother's Milk

Ideally, lead transfer to human milk would include estimates of lead in ambient air and major sources of oral exposure over time along with human milk estimates from the exposed lactating population. However, few studies have attempted to correlate lead exposure from multiple pathways (e.g. oral sources such as contaminated food, water, dust and soil and inhalation sources such as ambient air) with lead concentrations in human mother's milk. This is likely due to the multiple effects of daily intake from environmental sources (Sannolo et al., 1995) and internal transfer from lead released from bone stores during pregnancy and lactation (Gulson et al., 1997).

Although exposure to lead can come from many sources, ambient air contaminated from combustion sources has been a significant source of exposure in the U.S. population and European countries (U.S. EPA 1998). The relationship between air lead and blood lead has been studied extensively in both field studies and experimental chamber studies. OEHHA evaluated studies conducted prior to 1997 in their health risk assessment of inorganic lead under the toxic air contaminant program (OEHHA, 1997).

Briefly, in the OEHHA report, the contribution of airborne lead to blood lead levels was examined using several different methods – disaggregate, aggregate, uptake biokinetic, and physiologically based pharmaco-kinetic models (OEHHA, 1997). Findings were evaluated for linearity over a wide range of air and blood lead levels and are expected to apply to some exposure scenarios under the Hot Spots program. Most of these studies were conducted prior to 1985 when both air and blood lead levels were much higher than they are now. For example, the level of lead in the air used in chamber studies was 3.2 $\mu g/m^3$ representing low exposure and 10.9 $\mu g/m^3$ representing high exposure, while background air was typically between 7 $\mu g/m^3$ and 8 $\mu g/m^3$ in the city of Los Angeles during similar time-periods – late 1960s / early 1970s. Lead in Los Angeles air is 100-fold lower today (Ospital et al., 2008).

The relationship between air lead concentration and blood lead is not linear. Higher slopes are observed at lower air lead concentrations. However, the aggregate model was chosen because it implicitly incorporates all air-related pathways (i.e. soil, dust, water, contaminated food, etc.) and has averaged slopes estimated from a wide range of air concentrations. Using this model OEHHA estimated that an average change of 1.8 µg/dL in adult blood lead levels ($\mu g/m^3$) per $\mu g/m^3$ air lead concentration with current ambient air levels in California

As part of our effort to estimate a lactational transfer factor for lead (Tco), we searched for studies that examined slope factors in other populations or were conducted subsequent to our 1997 report (OEHHA, 1997).

In addition to the kinetics of lead in the general adult population, recent studies have observed that - under similar exposure conditions - plasma lead rises by about 20% – 80% during lactation (Gulson et al., 1997; Gulson et al., 1998b; Gulson et al., 1999; Rothenberg et al., 2000; Tellez-Rojo et al., 2002). Findings from these and other investigations suggest that, in addition to daily environmental sources of exposure, breast milk levels of lead also reflect lead released from lead accumulated in the lactating woman's bones.

We were not able to locate studies that measured both long-term exposure to ambient air lead and lead levels in breast milk. Therefore, we calculated estimates of transfer from blood to human milk from separate study populations to combine with estimates of lead transfer from air to blood.

J.4.4 Population Transfer Coefficient (Tco) for Lead

OEHHA has derived transfer coefficients for lead using Equation J-9

$$Tco_{hma}=(Cma/C_{blood}^{+})x(C_{blood}^{+}/C_{blood})x(C_{blood}/(C_{air} \times BR))xF_{c1}\,xF_{c2} \quad \text{Eq. J-9}$$

where:

Cma = geometric mean human milk lead level (µg/L-milk as wet weight)
C_{blood}^{+} = geometric mean blood lead level during lactation (µg/dL)
C_{blood} = geometric mean blood lead level during non-lactating state (µg/dL)
C_{air} = geometric mean concentration of lead in ambient air (µg/m^3)
BR = geometric mean breathing rate for adult women (14 m^3/day)
F_{c1} = conversion factor (L-milk)/(kg-milk) ~ **(**0.97**)**
F_{c2} = conversion factor (dL)/(L) = 10

Cm_a is the geometric mean human milk lead level that incorporates all (aggregated) air-related pathways of lead. C_{blood}^{+} is the geometric mean blood lead level among lactating women measured during lactation (µg/L). C_{blood} is the geometric mean blood lead level taken from the general population during a non-lactating state (µg/L). C_{air} is the geometric mean concentration of lead in the ambient air (µg/m^3) inhaled by the same population where blood lead levels were measured. BR is the geometric mean breathing rate for adult women (14 m^3/day) (see Chapter 2). F_{c1} is the inverse of the specific gravity of breast milk (1.03 g/ml)(Sergen, 2006). F_{c2} is the conversion from deciliters to liters.

*J.4.4.1 **Biotransfer from Blood to Milk***

Three groups measured maternal blood lead before and during lactation along with lead in mother's milk (Gulson et al., 1997; Gulson et al., 1998a; Gulson et al., 1998b; Sowers et al., 2002; Ettinger et al., 2004). However, Sowers et al. reported unusually high levels of lead in breast milk relative to blood, which suggest contamination problems. It is possible that breast milk samples were contaminated by the sampling collection technique (e.g. lead in the nipple shields). However, it is also possible that a more efficient active transport mechanism at lower blood lead levels could explain higher levels of lead in breast milk relative to blood. More studies of mothers with low blood lead levels are needed to further verify the results reported by Sowers et al.

For our purposes, Gulson et al (1995, 1997, 1998a, 1998b) and Ettinger et al (2004) provide the best estimates of the change in blood lead levels before the onset of lactation, during lactation and relative to the levels of lead in breast milk (Gulson et al., 1997; Gulson et al., 1998a; Gulson et al., 1998b; Ettinger et al., 2004).

*J.4.4.2 **Transfer from Air to Blood***

Equation J-10 describes estimation of aggregate transfer from airborne and associated sources that appears in the OEHHA 1997 report on the health effects of airborne inorganic lead (OEHHA, 1997):

Slope factor = $(C_{bloode} - C_{bloodr})/(C_{aire} - C_{airr})$ Eq.-J-10

$(C_{bloode} - C_{bloodr})$ is the difference between lead concentration in the blood of exposed compared to reference group and $(C_{aire} - C_{airr})$ is the difference in air lead between exposed and reference group. This simplified model assumes that the exposed and reference communities are similar in confounders such as age and smoking habits and reasonably comparable in their exposure to other sources of lead (e.g. paint).

Subsequent to OEHHA's 1997 report, Ranft et al (2008) published results from studies conducted on exposure to air pollutants among residents living near industrial sources along the rivers Rhine, Ruhr and Wupper in North Rhine-Westphalia Germany during five time-periods from 1983 to 2000. Authors reported the distribution of ambient air lead levels for each of the five time-periods (Ranft et al., 2008).

During the early years (1983 – 1991), ambient air lead levels ranged from 0.100 – 0.510 µg/m^3. Whereas, during the later years (1997 – 2000), air lead levels were much more variable - ranging from 0.025 to 0.729 µg/m^3. The 50^{th} percentile (P 50) declined by almost a factor of 20 from years 1983 to 2000. During the earliest years (1983 – 1991), P 50 declined by a factor of four from 0.465 to 0.100 µg/m^3. Based on data collected from 1991 to 2000, these investigators reported that childhood blood lead would decrease by a factor of 6.4: 95%CI (6.02 – 6.80) from the decrease in lead concentration in polluted ambient air (m^3/dL).

OEHHA calculated a similar slope factor from the study of 500, 55-yr-old women living in industrial areas of the North Rhine – Westphalia, Germany from 1985 to 1990 by Wilhelm and associates (Wilhelm et al., 2007). The investigators reported that mean blood lead levels among these women declined from 7.2 to 5.0 µg/dL. Based on ambient air levels of lead reported in Ranft et al (2008), OEHHA estimated that blood lead levels in 55-year old women would change by 6-fold per unit of change in ambient air levels of lead (µg/dL) over a similar period (GM, 6.2; 95% CI 6.1 – 6.4) (Ranft et al., 2008). This estimate is within the range of slope factors reported previously by OEHHA for the general adult population (OEHHA, 1997).

J.4.3.3 Transfer from Air and Body Stores to Milk

Tables J.4-4 and J.4-5 show the Tcos derived by combining air to blood and blood to milk transfer of inorganic lead from the available data. Table J.4-4 shows the transfer factors derived from the study of eight women who provided samples of blood before and during lactation as well as samples of milk during lactation (Gulson et al., 1998a; Gulson et al., 1998b). The geometric mean and standard deviation blood lead levels prior to lactation were low (GM 2.2 µg/dL, GSD1.3).

Table J.4-4: Transfer Coefficients (Tcos) for Inorganic Lead Measured in Human Blood and Milk (d/kg-milk) from Data Reported in (Gulson et al., 1998a; Gulson et al., 1998b) and the Change in Blood Lead with the Change in Lead Concentration Measured in Ambient Air (slope factor)

Source	Slope factor m^3/dL	Tco (d/kg milk) GM	GSD	LCL	UCL
OEHHA	1.8	0.024	3.19	0.009	0.061
Willhelm/Ranft	6.2	0.08	3.19	0.031	0.203

LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

Table J.4-5 shows the transfer factors derived from the study of 253 women who provided samples of blood prior-to and during lactation as well as samples of milk during lactation (Ettinger et al., 2004).

Table J.4-5: Biotransfer Coefficients (Tcos) for Inorganic Lead Measured in Human Blood and Milk (d/kg-milk) from Data Reported in (Ettinger et al., 2004) and the Change in Blood Lead with the Change in Lead Concentration Measured in Ambient Air (slope factor)

Source	Slope factor m^3/dL	Tco (d/kg milk) GM	GSD	LCL	UCL
OEHHA	1.8	0.019	3.00	0.017	0.022
Willhelm/Ranft	6.2	0.064	3.00	0.056	0.074

LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

Compared to Gulson et al (1998), the geometric mean, blood lead levels prior to lactation observed by Ettinger et al (2004) were about 4-fold higher (7.3 and 8.0 for exclusive and partial lactators, respectively)(Gulson et al., 1998b; Ettinger et al., 2004). However, the transfer factors derived from residents of Mexico and immigrants to Australia differ by less than a factor of two.

J.4.4 Study Limitations, Influencing Factors and Uncertainty (inorganic compounds)

Our Tco estimate for lead has not considered the influence of maternal age, parity, length of lactation, and body weight on concentration of lead in milk.

J.5 Summary and Recommendations

This appendix develops lactational transfer coefficients for use in estimating the concentration of a multipathway chemical in mother's milk from an estimate of chronic incremental daily dose to the mother from local stationary sources. OEHHA derived human lactational transfer coefficients from studies that measured contaminants in human milk and daily intake from inhalation or oral exposure (e.g. air, cigarette smoke or diet) in the same or a similar human population. These coefficients can be applied to the mother's chronic daily dose estimated by the Hot Spots exposure model to estimate a chemical concentration in her milk.

We established transfer coefficients (Tcos) for individual congeners and WHO-TEQ summary PCDDs/Fs and dioxin-like-PCBs, individual and summary carcinogenic PAHs, and lead through equations J-1-3, data on exposure and breast milk contamination from background (global), accidental and occupational sources, and a set of simplifying assumptions. We assume that a mother's intake and elimination is constant before lactation. We also assume that changes in a woman's body due to the onset of lactation occur as a single shift in elimination rate over the lactation period. In some cases, OEHHA adjusted some measurements of human milk and contaminant intake to account for confounding factors. In such cases, OEHHA describes the method of adjustment in the text and table containing adjusted values.

We described the methods for deriving specific Tcos from measurements of human milk, intake and transfer estimates from studies of populations exposed to general global sources of pollutants. Although the proportional contribution from various exposure pathways to total exposure from a single Hot Spots facility is likely to be quite different from exposure found with global sources, we believe Tcos in this appendix have been derived from data that serve as reasonable surrogates of transfer from Hot Spot facility exposures.

J.5.1 Dioxins and Furans

Personal factors such as body fat, smoking status and past lactation practices can affect body burden and elimination rates. For example, smoking has been associated with a 30% to 100% increase in elimination rates of some dioxin congeners (Milbrath et al. 2009, Flesch-Janys et al. 1996). As well, the onset of lactation sets a new elimination pathway into effect and can substantially reduce the maternal body burden of PCBs during 6 months of lactation (Niessen et al.1984, Landrigan et al. 2002).

Therefore, OEHHA incorporated conservative assumptions regarding these factors into our model (i.e. reference half-lives based on body burden below 700 ppt in the blood, adult age, nonsmoker, no recent prior breast-feeding period and

percent body fat of older adults) in addition to accounting for the substantial variability between individual congeners of PCDDs, PCDFs and dioxin-like PCBs.

To calculate oral Tcos, OEHHA used adjusted reference half-lives for the chemicals in the adult human body derived from dietary and occupational exposures. OEHHA estimated oral Tcos for these chemicals from estimates of body weight reported in Chapter 10 of this document, the steady-state equation developed by Smith (1987) and reference half-lives reported in Milbrath et al (2009). Milbrath et al (2009) adjusted reference half-lives for age, body fat, smoking habits and breast-feeding status as these factors were all strong determinants of half-life in humans.

A carryover rate > 1 would suggest that dioxins and dioxin-like compounds could accumulate in body fat and transfer to the fat in mother's milk. An average dioxin Tco of 3.7 d/kg indicates that 370% of the daily intake from ingested sources transfers to mother's milk. This high transfer-value suggests that some accumulation of carcinogenic dioxins and dioxin-like compounds occurs in the mother's body. For individual congeners, an oral Tco less than one (e.g. 1,2,3,4,7,8-HxCDF and 2,3,4,6,7,8-HxCDF) suggests that some metabolism occurs in the mother's body.

J.5.2 PAHs

Based on the estimated intake of 16 measured PAHs in simulated smoking studies and the PAHs found in breast milk from long-time smoking mothers (Zanieri et al. 2007), OEHHA was able to estimate transfer coefficients (Tco) with a modified version of Equation J-1.

The key assumption underlying the development of these Tcos is that the variability in an individual PAHs Tcos is sufficiently small to justify the use of an average value for individual PAH congeners. This approach appears to be the best available given the available studies.

OEHHA calculated oral Tcos for each individual PAH by Equation J-8. The average Tco for carcinogenic and PAHs without cancer potency factors was calculated as the sum of the Tco values over the total number of PAHs evaluated. Similar Tco values are obtained for both groups of PAHs (0.46 d/kg) and 0.31 d/kg, respectively). This finding suggests that, on average, the PAHs with cancer potency factors as a whole transfer to mother's milk with about the same efficiency as some of the most common PAHs without cancer potency factors that are taken in through the diet. Therefore, summary Tcos were calculated by pooling across individual PAH-Tcos from both groups (see Table J.3-7).

J.5.3 Inorganic Lead

In an effort to derive lactational transfer coefficients for inorganic lead, OEHHA has drawn from studies conducted on subjects exposed to lead through multiple pathways at higher levels from other areas of the world. OEHHA assumes that the transfer of lead derived from these studies serves as a reasonable surrogate for the transfer of lead from contaminated media near a Hot Spots facility in California.

We were not able to locate studies that measured both long-term exposure to ambient air lead and lead levels in breast milk. Therefore, we calculated estimates of transfer from blood to human milk from separate study populations to combine with estimates of lead transfer from air to blood.

For our purposes, Gulson et al (1995, 1997, 1998a, 1998b) and Ettinger et al (2004) provide the best estimates of the change in blood lead levels due to the onset of lactation as well as during lactation relative to the levels of lead in breast milk.

Based on ambient air levels of lead reported in Ranft et al (2008), OEHHA estimated that blood lead levels in 55-year old women would change by 6-fold per unit of change in ambient air levels of lead (µg/dL) over a similar period (GM, 6.2; 95% CL 6.1 – 6.4).

Compared to Gulson et al (1998), the geometric mean blood lead levels prior to lactation observed by Ettinger et al (2004) were about 4-fold higher (7.3 and 8.0 for exclusive and partial lactators, respectively) (Gulson et al., 1998b; Ettinger et al., 2004).

The transfer factors derived from residents of Mexico and immigrants to Australia differ by less than a factor of two. However, our Tco estimate for lead has not considered the influence of maternal age, parity, length of lactation, and body weight on concentration of lead in milk.

J.5.4 Recommendations

OEHHA recommends using the Tcos based on the summary estimates provided in Table J.1-1 rather than the individual compound Tcos provided in Tables J.2-3, J.3-4, and J.3-6 to assess transfer of compounds to mother's milk. Tcos of individual compound are less robust that summary Tcos listed in Table J.1-1 because in some cases they have derived from data containing a high number of non-detects and small sample sizes. Additional studies might improve the estimation of individual Tco values, especially studies that incorporate more sensitive methods for analyzing breast milk PAH content and larger study populations to better estimate biological variation and estimates of PAH transfer

from air to mother's milk. Such improved data could allow for a robust determination of the Tco values for individual compounds (see Table J.1-1).

Table J.1-1: Default Tcos (d/kg) for Mother's Milk

Chemical/chem. group	Tco	LCL	UCL
PCDDs - oral	3.7	2.68	5.23
PCDFs - oral	1.8	1.27	2.43
Dioxin-like PCBs - oral	1.7	0.69	4.40
PAHs – inhalation	1.55	0.731	3.281
PAHs – oral	0.401	0.132	1.218
Lead - inhalation	0.064	0.056	0.074

LCL, lower 95% confidence interval of the mean Tco; UCL, upper 95% confidence interval of the mean Tco

When calculating cancer risk from speciated PCDD/Fs, dioxin-like PCBs and PAHs, assume that the ratios of congeners measured in the emissions are preserved when transferred from the mother's body to breast milk. OEHHA recommends a single Tco for each chemical group (e.g. PCDDs oral). Risk assessors can apply TEQs to the infant dose after applying the Tco for a chemical group to each congener in the group to calculate infant cancer risk for the mother's milk pathway.

The mother's exposure from multiple pathways should be included in estimating the concentration of contaminant in mother's milk. One key factor that plays a role in the difference between oral and inhalation transfer coefficient (e.g., for PAHs) is first pass metabolism which is lacking in dermal and inhalation exposures. Thus, for simplicity, OEHHA applies the transfer coefficients from inhalation to the dermal absorption pathway for lead and PAHs. For lead, we are using the inhalation Tco for all the other pathways of exposure to the mother. Likewise for PCDD/Fs and dioxin-like PCBs, we are using the oral Tco for the other pathways of exposure to the mother in Eq. J-2.

J.6 References

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